Inquiry into the convictions of Kathleen Megan Folbigg

PART 3

CHAPTER 7: MEDICAL EVIDENCE

Caleb

Overview of evidence on cause of death

1. Dr Cummings found on his autopsy of Caleb that the lungs were moderately moist,[1] and the histology recorded they were congested and in places showed incomplete aeration.[2] Some alveoli contained extravasated red blood cells and a small amount of eosinophilic exudate.[3] These were all non-specific findings.[4] Microscopic examination showed congestive changes with focal areas of haemorrhage with some alveolar spaces.[5] These were very common and again, non-specific findings.[6]

2. None of these features enabled a forensic pathologist to identify a cause of death.[7] Nor were any of them specifically suggestive that Caleb had been smothered.[8]

3. While there was no histology of Caleb's upper airway, Professor Berry reported that Caleb's lungs showed no evidence of infection.[9] Similarly, Professor Busuttil noted in his report that the autopsy failed to reveal any infection.[10]

[1] Exhibit H, Forensic pathology tender bundle, p 10.
[2] Exhibit H, Forensic pathology tender bundle, p 10.
[3] Exhibit H, Forensic pathology tender bundle, p 10.
[4] Transcript of the Inquiry, 19 March 2019 T126.49-T127.10.
[5] Exhibit H, Forensic pathology tender bundle, p 23.
[6] Transcript of the Inquiry, 19 March 2019 T124.14; T124.20-21; T124.31-36.
[7] Transcript of the Inquiry, 19 March 2019 T129.1-T132.6; T139.20-33.
[8] Transcript of the Inquiry, 19 March 2019 T127.21-T129.28.
[9] Exhibit H, Forensic pathology tender bundle, p 251.

4. In the Inquiry, Dr Cala referred to the presence of blood and froth around Caleb's mouth as a sign of concern.[11] This was identified in the report of Caleb's death to the coroner.[12] Dr Cala observed that the report suggests it was a small amount, but the volume is unknown.[13] Dr Cala said it does not exclude the possibility of some external agent having been applied to Caleb's outer airway, whether accidentally or deliberately, particularly given Caleb was in a supine position when found, and decomposition was not a possible cause.[14]

5. The other forensic pathologists did not share Dr Cala's views regarding the blood and froth. Professor Duflou said that in the absence of anything else, it did not negate SIDS as an entirely reasonable cause of death.[15] In the Inquiry, and also at trial, Professor Hilton described frothy, bloody fluid at the nose and sometimes mouth as commonplace in SIDS. Professor Cordner opined that such a finding was not particularly exceptional in SIDS.[16] These views are consistent with Professor Berry's evidence at trial, who said that blood-stained froth is a common finding in SIDS and in suffocation.[17]

6. In our submission, the weight of the expert evidence is that the blood and froth could have been present whether Caleb's death was SIDS or the result of accidental or deliberate smothering.

7. Before the trial, Professor Berry was provided with 14 stained microscope slides of tissue taken from Caleb's body.[18] He said that some time subsequent to the death, sections of lung had been stained by Perl's method for ferric iron.[19] It was believed that some children, who experience a period of complete occlusion of the airways and recover, bleed into their lungs.[20] Over a period of 36 to 48 hours the blood is converted into haemosiderin, which stains blue via the Perl's method.[21] One of

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[10] Exhibit H, Forensic pathology tender bundle, p 308.
[11] Transcript of the Inquiry, 19 March 2019 T127.25-26.
[12] Exhibit H, Forensic pathology tender bundle, p 4; Transcript of the Inquiry 19 March 2019 T128.25-27.
[13] Transcript of the Inquiry 19 March 2019 T128.5-8.
[14] Transcript of the Inquiry 19 March 2019 T128.36-47.
[15] Transcript of the Inquiry 19 March 2019 T130.3-10.
[16] Transcript of the Inquiry 19 March 2019 T130.18-20; 14 April 2003 T632.46-48.
[17] Exhibit H, Forensic pathology tender bundle, p 254.
[18] Exhibit H, Forensic pathology tender bundle, p 251.
[19] 1 May 2003 T1058.32-36.
[20] 1 May 2003 T1057.55-57.
[21] 1 May 2003 T1057-57-T1058.2.

the explanations for a positive Perl's stain is that there may have been an episode of previous asphyxia, whatever the cause.[22]

8. Professor Berry saw a significant amount of haemosiderin on sections of Caleb's lungs, in the tissue and the air spaces.[23] Presence in the air space was linked to suffocation.[24] Professor Berry said that haemosiderin is a very unusual finding in SIDS and would prompt investigation into the possibility of a previous episode of suffocation.[25]

9. However, Professor Byard gave evidence that he had done a study in which he found haemosiderin in around 20 per cent of SIDS babies' lungs, and it just meant that something had happened with bleeding in the past which could be suffocation or could, for example, be a nose bleed.[26] No one knows how much iron would get into the lungs from child birth; any inhalation of blood into lungs could cause iron presence.[27] This view is also expressed in Duncan and Byard (2018).[28] Professor Byard also said that one of the most common causes of bleeding from *within* the lungs is an asphyxiating event of some sort, and it possibly tended to indicate a previous episode of this type in Caleb.[29]

10. The Inquiry was informed that the slides seen by Professor Berry are not now available. When and by whom they were stained is not known.

11. In the Inquiry, again consistently with Duncan and Byard (2018),[30] the forensic pathologists said that the view today is that haemosiderin in the lungs is not a positive indicator of superimposed upper airway obstruction.[31] It is still understood that it takes a number of days after the blood is deposited for haemosiderin to become apparent.[32] There is no evidence of where it came from in Caleb.

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[22] 1 May 2003 T1058.2-4.
[23] 1 May 2003 T1059.1-4.
[24] 1 May 2003 T1059.47-57; T1060.16-23.
[25] 1 May 2003 T1059.10-14; T1060.10-14.
[26] 7 May 2003 T1208.23-35.
[27] 7 May 2003 T1208.26-35.
[28] Exhibit D, Jhodie R Duncan and Roger W Byard (eds), *SIDS: Sudden Infant and Early Childhood Death – The Past, the Present and the Future* (University of Adelaide Press, 2018) 504-505.
[29] 7 May 2003 T1235.13-24.
[30] Exhibit D, Jhodie R Duncan and Roger W Byard (eds), *SIDS: Sudden Infant and Early Childhood Death – The Past, the Present and the Future* (University of Adelaide Press, 2018) 504-505.
[31] Transcript of the Inquiry, 20 March 2019 T232.30-39.
[32] Transcript of the Inquiry, 20 March 2019 T233.16-34; Exhibit D, Jhodie R Duncan and Roger W Byard (eds), *SIDS: Sudden Infant and Early Childhood Death – The Past, the Present and the Future* (University of Adelaide Press, 2018) 504-505.

SIDS

12. Caleb's death was recorded as SIDS by Dr Cummings, who conducted the autopsy, and on the death certificate.[33]

13. At trial, Dr Beal said that she would have given SIDS as a cause of Caleb's death on its own, albeit this was unlikely given he was found supine, and with a proviso that he was under three weeks old.[34] However, Dr Cala and Professors Herdson, Byard and Busuttil would have given the cause of death as undetermined.[35] Dr Cala said a lack of positive finding of suffocation did not exclude suffocation,[36] and Professor Herdson said it was apparently consistent with SIDS but he could not distinguish between SIDS and suffocation.[37] Professor Byard would not have excluded SIDS, his preference for undetermined being in view of the laryngomalacia, or floppy larynx and because there was no death scene investigation, no histology of the brain and a history of breathing problems with diagnosis of floppy larynx.[38] Professor Berry would have excluded SIDS in view of the finding of haemosiderin.[39]

14. No expert at trial had heard of a child who had died from floppy larynx. Dr Beal and Professor Byard both said it was most unlikely to have played a role. Professor Herdson said it did not indicate inflammation or infection,[40] and Professor Berry explained it is generally a benign and self-limiting condition – not a recognised cause of death.[41]

15. In the Inquiry, Professors Duflou, Cordner and Hilton would all have given Caleb's death as Category 2 SIDS, all because of the presence of laryngomalacia. Caleb falls within Category 2, being younger in age than the classic SIDS range.

16. Professor Duflou noted research that 90 per cent of laryngomalacia is benign and self-limiting, echoing Professor Berry at trial, but noted that the remainder if

[33] Exhibit H, Forensic pathology tender bundle, pp 3, 9.
[34] 5 May 2003 T1138.23-40.
[35] 16 April 2003 T746.48-53; Exhibit H, Forensic pathology tender bundle, p 274; 7 May 2003 T1202.17-26, T1209.6-9; Forensic pathology tender bundle p 279 (Professor Byard in his report stated he would have noted a history of breathing problems involving a floppy larynx (laryngomalacia)).
[36] 15 April 2003, T730.33-35.
[37] Exhibit H, Forensic pathology tender bundle, p 274.
[38] 7 May 2003 T1209.9-24.
[39] 1 May 2003 T1059.10-14.
[40] 1 May 2003 T1034.20-27.
[41] 1 May 2003 T1057.11-13.

untreated can prove fatal.[42] Professor Hilton agreed, saying that it could not be proved or disproved whether laryngomalacia played a part.[43] Professor Cordner considered that laryngomalacia potentially meant that Caleb was more vulnerable to SIDS.[44]

17. In the Inquiry, Dr Cala tempered his view at trial, where he said that he considered that Caleb was "likely smothered". He maintained that he would ascribe Caleb's death as undetermined, in view of Caleb's age and concern about the report of blood and froth.[45] Dr Cala did not accept the floppy larynx was a cause of death; he was not convinced laryngomalacia was in any way serious.[46]

Time of death

18. Professor Duflou observed that the time of Caleb's death was not formally assessed at autopsy or by ambulance officers, but noted Caleb was described as cold to touch by Ms Folbigg, and either warm or cold to touch by ambulance officers.[47]

19. Professor Duflou also observed Caleb to have a large quantity of curdled milk in his stomach at the time of autopsy, noting that the time taken for the stomach to empty in infants is variable and complex, but in general one to two hours is not unreasonable.[48] He acknowledged, however, that providing opinions based on stomach content is dangerous for forensic pathologists, and there are "probably graveyards full of forensic pathologists who have done that" – Professor Cordner described it as "tiger country".[49] Professor Duflou stated it appeared that Caleb died some short time after he was checked by his mother at 10:00pm, and likely not around the time he was next checked by her at 2:45am.[50]

20. In a statement to police, Ms Folbigg stated that she fed Caleb at 1am then put him to bed.[51] She recorded in a diary, at about 2:00am, that Caleb was "finally asleep".[52] At about 2:50am, she checked him and found him cold.[53] The first unit

[42] Transcript of the Inquiry, 21 March 2019 T245.27-31.
[43] Transcript of the Inquiry, 21 March 2019 T278.4-6.
[44] Transcript of the Inquiry, 21 March 2019 T278.12-16.
[45] Transcript of the Inquiry, 19 March 2019 T129.1-40.
[46] Transcript of the Inquiry, 21 March 2019 T277.34-41.
[47] Transcript of the Inquiry, 19 March 2019 T141.39-40.
[48] Exhibit L, Expert report of Professor Johan Duflou (13 February 2019) p 29.
[49] Transcript of the Inquiry, 19 March 2019 T125.40-44, T126.10.
[50] Exhibit L, Expert report of Professor Johan Duflou (13 February 2019) p 29.
[51] Exhibit AZ, Diaries tender bundle, p 30.
[52] Exhibit AZ, Diaries tender bundle, p 19.

to arrive at 2:59am found Caleb was unconscious, not breathing and pulseless, and warm to touch.[54]

21. Mr Reed was an ambulance officer in the second unit to arrive. That unit used an ECG upon their arrival.[55] Mr Reed described Caleb's skin temperature as cold to touch, and obviously not breathing and with no circulation.[56] He also recorded the airway as "clear", whereas the first officers recorded the airway as "obstructed", which may indicate there was some time period between the two, although Mr Reed recorded his arrival time as 3:03am.[57]

22. Professor Duflou agreed that the body temperature described by ambulance officers was not known, and also said that an assessment of body temperature on the basis of feel is very unhelpful.[58] He said he based his opinion predominantly on the stomach contents. Professor Hilton doubted that these observations had any relevance as to when Caleb died.[59] He agreed that it was obvious that if Caleb was fed at 1:00am, or early in the morning, and finally went to sleep at 2:00am, death could not have occurred prior to 2:00am.[60]

23. In light of Caleb having been fed at around 1:00am, and of the ambulance officers' evidence, Professor Duflou's statement that Caleb likely died around 10:00pm and likely not around 2:45am should not be accepted.

Submissions on cause of Caleb's death

24. There have been two material changes since the 2003 trial. First, genetic testing has been completed and no genetic variant which is pathogenic or likely pathogenic has been identified to account for Caleb's death (or any of his siblings). Secondly, more recent research on SIDS that maternal smoking and sleeping position pose the highest risks relevantly reduces any assessment of Caleb's risk of SIDS.

25. In other respects, the opinions expressed to the Inquiry, based on the same information available in 2003, remain broadly similar to those given at the trial.

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[53] Exhibit AZ, Diaries tender bundle, p 30.
[54] Exhibit H, Forensic pathology tender bundle, pp 15, 21; 3 April 2003 T142.27-28.
[55] Exhibit H, Forensic pathology tender bundle, p 13.
[56] Exhibit H, Forensic pathology tender bundle, p 13.
[57] Exhibit H, Forensic pathology tender bundle, p 18.
[58] Transcript of the Inquiry, 19 March 2019 T142.4.
[59] Transcript of the Inquiry, 19 March 2019 T121.46.
[60] Transcript of the Inquiry, 20 March 2019 T145.19-20.

26. It remains the case that any contribution to Caleb's death by laryngomalacia is highly unlikely.

27. The weight of the expert opinion at trial was that his death was best described as undetermined. In the Inquiry, apart from Dr Cala the forensic pathology experts preferred Category 2 SIDS.

28. Whereas experts at trial hesitated or qualified ascribing SIDS in large part due to Caleb being 19 days old when he died, and younger than generally acceptable SIDS age at the time, Category 2 SIDS expressly contemplates the death of a child Caleb's age. In the Inquiry Professor Duflou said, Professor Hilton agreeing, that Caleb's death ought to be ascribed as SIDS 2 *because of* his age at time of death.[61]

29. However described, SIDS – and particularly Category 2 SIDS – does not answer the cause of Caleb's death. SIDS does not exclude unidentified natural causes. Nor does SIDS, and particularly Category 2 SIDS, described as being more flexible, exclude unnatural causes which are not identifiable at autopsy. Finally, it can be virtually impossible to distinguish at autopsy between a SIDS death and a death caused by deliberate or accidental suffocation.

30. On forensic pathology evidence, both "undetermined" and SIDS apply to Caleb's death. Both terms leave open the possibility of an unidentified natural cause, or unidentified unnatural cause, of death.

31. Ultimately, on the medical evidence in 2019 there remains no identified natural (including genetic) cause of Caleb's death and death from unnatural causes cannot be excluded.

32. Most medical experts considered that the death could have been the result of an asphyxiating event. No medical expert excluded asphyxia or smothering.

[61] Transcript of the Inquiry, 19 March 2019 T130.9-20.

Patrick's ALTE

Overview of evidence on cause of ALTE

33. Mr Hopkins, an ambulance officer who attended when Patrick suffered the ALTE, described Patrick as appearing to be having respiratory difficulties, pale and listless, and exhibiting tracheal tug and intercostal recession.[62] He administered oxygen therapy en route to the hospital, in response to which Patrick's level of consciousness rose although his respiratory effect remained impaired.[63] Patrick presented at the hospital with an oxygen saturation level of 88%.[64]

34. Dr Joseph Dezordi was a neonatal paediatric consultant who examined Patrick when Patrick was brought to the Mater Hospital.[65] He described Patrick as being hypoxic – blue, lethargic, with no fever.[66] The initial treatment included administering oxygen.[67] Patrick's colour and oxygen saturation level normalised fairly rapidly over 15 to 20 minutes. Because of that, Dr Dezordi concluded that he was not dealing primarily with pathology involving his lungs, chest or possibly airways such as pneumonia or bronchiolitis.[68] Patrick's condition was also not likely to be due to a respiratory problem because he remained pink even when a high concentration oxygen was not being administered.[69] Virological tests did not support bronchiolitis.[70]

35. Patrick was arching his back at times, which Dr Dezordi said was non-specific although it could have indicated inflammation of the brain.[71] After investigations, there were no signs of serious illness such as meningitis, septicaemia or meningococcal, or trauma or injury.[72] A urine test returned unexpected high glucose, suggesting an asphyxiating event – being any event that leads to

[62] Exhibit H, Forensic pathology tender bundle, p 17.
[63] Exhibit H, Forensic pathology tender bundle, p 17; 9 April 2003 T436.48-50.
[64] Exhibit S, Section of Patrick's medical records, p 534.
[65] 9 April 2003 T446.8-T447.13; Forensic pathology tender bundle, p 74.
[66] 9 April 2003 T446.46-447.13; T452.8-13.
[67] 9 April 2003 T447.55.
[68] 9 April 2003 T448.2-41; Exhibit H, Forensic pathology tender bundle, p 76.
[69] Exhibit H, Forensic pathology tender bundle, p 75.
[70] Exhibit H, Forensic pathology tender bundle, p 76.
[71] 9 April 2003 T482.49-T483.4.
[72] 9 April 2003 T450.5-25.

obstruction of air into the lungs and impairment of oxygen levels in the blood and to the brain or prolonged seizure.[73]

36. An EEG on 18 October 1990 indicated normal functioning.[74] However, on the evening of 19 October 1990, Patrick developed recurrent seizures.[75] A second EEG of 5 November 1990 showed abnormalities, which in evidence Dr Dezordi said would not necessarily have been pathognomonic of encephalitis, and could have been a process of untreated or unresolved epilepsy.[76] An encephalopathic process is much broader than encephalitis and could be due, for example, to an ongoing infection, metabolic disease, or brain damage due to hypoxia.[77] Thus, the abnormalities which showed on 5 November 1990 were not necessarily pathognomonic of encephalitis and in any event, by 5 November 1990 any diagnosis of herpes encephalitis would have been tenuous. In the interim, normal lumbar punctures had been conducted, which were a far more powerful test for encephalitis.[78]

37. The report of a CT scan taken on 23 October 1990 indicated some brain abnormalities in the occipital lobes (at the back), and temporal lobes (at the side).[79] In light of a normal EEG, lumbar puncture and tests for viruses, the causes of the abnormality in Patrick's brain and seizures were unknown. His fits were stabilised with anticonvulsants and Patrick was discharged with a diagnosis of intractable seizures, probably viral encephalitis and bronchiolitis.[80]

38. On 4 November 1990 Patrick presented with prolonged seizures which resolved spontaneously after 90 minutes. An EEG showed multifocal epileptogenic foci suggesting a progressive encephalopathic disorder. A repeat CT scan on 5 November 1990 showed deterioration, and damage at the back of Patrick's brain.[81] Investigations after a further admission on 14 November 1990 showed with a query, an occipital ischaemic area with clinical visual impairment (probably

[73] 9 April 2003 T449.38-450.3, T458.35-46; Exhibit H, Forensic pathology tender bundle, p 75.
[74] 9 April 2003 T458.35-46.
[75] 9 April 2003 T466.45-56; Exhibit H, Forensic pathology tender bundle, p 77.
[76] 9 April 2003 T460.11-31.
[77] 9 April 2003 T460.33-53.
[78] 9 April 2003 T461.1-11.
[79] 9 April 2003 T467.52-T468.6; 10 April 2003 T497.1-19; Exhibit H, Forensic pathology tender bundle, pp 72, 77.
[80] Exhibit H, Forensic pathology tender bundle, p 39.
[81] 9 April 2003 T469.43-55; 10 April 2003 T487.6-15.

cortical blindness) and developmental regression.[82] Other investigations including an echocardiography were negative.[83]

39. Patrick was admitted again on 22 December 1990 with an oculogyric crisis secondary to past encephalitic basal ganglia problem, provoked by a viral illness.[84]

40. At trial, Dr Dezordi considered encephalitis to almost not be possible, relying in part upon the normal EEG of 18 October 1990.[85] Dr Ian Wilkinson, a paediatric neurologist who saw Patrick in relation to the ALTE, absolutely excluded encephalitis in evidence.[86] Professor Byard gave evidence that he considered encephalitis was possible but he deferred to Dr Wilkinson and Dr Dezordi.[87]

41. Dr Wilkinson gave evidence that the damage to Patrick's brain was consistent with an asphyxiating event, because loss of visual function was seen in other cases of asphyxiating events in children, the visual part of the brain being extraordinarily sensitive to lack of oxygen.[88] He said it was not clearly understood why seizures would normally happen a few days after, but swelling in the brain may not reach maximum until the second, third or fourth day, and scarring may irritate normal electrical activity, producing seizures.[89]

42. Professor Byard considered the ALTE was most likely caused by an asphyxiating event.[90] Dr Beal agreed that the likely cause of the ALTE was an acute asphyxial event of undetermined origin.[91] In his report, Professor Ouvrier considered that an asphyxial event was the most plausible explanation, which could have been a near miss SIDS or suffocation, and that a pattern of delayed seizures was common in ALTEs of whatever cause.[92] Dr Cala at trial opined that the ALTE was possibly from a smothering or asphyxiating event.[93]

[82] Exhibit S, Section of Patrick's medical records p 774.
[83] Exhibit S, Section of Patrick's medical records p 748 T469.43-55; 10 April 2003 T487.6-15.
[83] Exhibit S, Section of Patrick's medical records p 774.
[84] Exhibit H, Forensic pathology tender bundle, p 39; Exhibit S, Section of Patrick's medical records p 780.
[85] 9 April 2003 T458.35-46.
[86] 10 April 2003 T517.18; T519.35, 23 April 2003 T859.57-860.1, T876.11.
[87] 7 May 2003 T1209.53-1210.3, T1236.19-1237.15, see T1213.35-56.
[88] 10 April 2003 T510.10-16.
[89] 10 April 2003 T510.27-41.
[90] 7 May 2003 T1209.56-T1210.7, T1209.51-52, T1212.35-45, T1237.32-38, T1254.54-57.
[91] 5 May 2003 T1138.55-T1139.12, T1147.35-36.
[92] Exhibit H, Forensic pathology tender bundle, p 286.
[93] 16 April 2003 T747.1-9.

43. All of the forensic pathologists who gave evidence in the Inquiry said that the cause of the ALTE was unknown or unexplained.[94] Dr Cala and Professor Hilton observed there was no indication of a degenerative neurological condition developing prior to 18 October 1990.[95]

44. It was put to the forensic pathologists in the Inquiry that they would not speculate whether Patrick's encephalopathic disorder developed on or prior to 18 October 1990. However, Dr Cala said that on the medical records and ambulance report, there was no information that Patrick was anything other than a well and normal child leading up to whatever caused the ALTE.[96] There did not appear to be any evidence of a chronic degenerative condition other than the infarcts and gliosis that were evident on brain examination after Patrick's death, which were not part of a chronic degenerative process.[97] If a chronic neurological condition triggered some epileptiform type disorder, Dr Cala would expect to see some pathological sign for the underlying degenerative condition on the EEGs (after the ALTE).[98] Dr Cala thought a degenerative neurological condition was highly unlikely, although he would defer to a paediatric neurologist.[99]

45. Two paediatric neurologists subsequently gave evidence in the Inquiry in relation to Patrick's ALTE and death. Professor Monique Ryan is a paediatric neurologist and Director of Neurology at the Royal Children's Hospital in Victoria. Associate Professor Michael Fahey is a paediatric neurologist, clinical geneticist and Director of Neurology at the Monash Children's Hospital in Victoria.

46. Professor Ryan was briefed by those representing Ms Folbigg and at the time of preparing her report had received some but not all of the clinical records concerning Patrick. On the basis of material she had received, Professor Ryan opined "I am not convinced that Patrick's clinical history is consistent with him having neurologic deficits resulting from a single hypoxic-ischaemic episode on October 18, 1990."[100]

[94] 20 March 2019 T146.1-T147.23; Exhibit L, Expert report of Professor Johan Duflou (13 February 2019) p 41; Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 60, fn 60, 90.
[95] Transcript of the Inquiry, 21 March 2019 T269.47-T270.
[96] Transcript of the Inquiry, 21 March 2019 T268.35-42.
[97] Transcript of the Inquiry, 21 March 2019 T269.3-9.
[98] Transcript of the Inquiry, 21 March 2019 T269.18-35.
[99] Transcript of the Inquiry, 21 March 2019 T269.47-T270.9.
[100] Exhibit AJ, Expert report of Professor Monique Ryan (15 March 2019) p 14.

47. The significance of this opinion is that if Patrick did not experience a single hypoxic-ischaemic episode on 18 October 1990, then his presentation was not consistent with him having been the subject of an attempted suffocation on that date.

48. Professor Ryan additionally did not accept that Patrick was a typically developing and otherwise well baby prior to 18 October 1990.[101] Professor Ryan pointed specifically to Patrick having had torticollis, which she accepted can be a benign phenomenon, and to Ms Folbigg's description, after his presentation, of him having always tended to arch his back at times. She said she did not know what to make of those factors, but they suggested a possibility that he was not entirely normal prior to 18 October 1990.[102]

49. No other expert took issue with the proposition that Patrick was a healthy and normally developing baby.[103] Dr Colley observed that torticollis is a not uncommon condition which is "often quite benign" and on its own does not make a diagnosis of a neurogenetic condition. She also observed that back arching is a common behaviour in healthy children when irritable. She opined it was hard in retrospect to know whether this was really relevant, and it would be more relevant if a treating practitioner had recorded prior to 18 October 1990 that Patrick was back arching.[104]

50. The basis for Professor Ryan's opinion that Patrick's presentation on 18 October 1990 was not consistent with a single hypoxic-ischaemic episode was the variability in Patrick's presentation from the time he presented at the hospital. In particular she considered that it was difficult to imagine he would have been able to feed well on 18 October 1990 and that his EEG would have been entirely normal, had he sustained a severe hypoxic-ischaemic insult sufficiently severe to cause the changes seen on his brain in subsequent imaging and at post-mortem examination.[105]

51. As to alternative diagnoses potentially causative of Patrick's ALTE and death, Professor Ryan explained that a number of conditions possibly associated with epilepsy and fluctuating neurologic symptoms were not excluded by previous

[101] Transcript of the Inquiry, 17 April 2019 T586.20-46.
[102] Transcript of the Inquiry, 17 April 2019 T585.44-46.
[103] The basis for which is set out above in Chapter 3: Health of the Folbigg children and Ms Folbigg.
[104] Transcript of the Inquiry, 17 April 2019 T591.35-50.
[105] Exhibit AJ, Expert report of Professor Monique Ryan (15 March 2019) p 14.

testing. She said those conditions include disorders of creatine metabolism, alternating hemiplegia of childhood, neurotransmitter disorders and genetic channelopathies causing infantile encephalopathies and cardiac arrhythmias.[106] She said in her report that further testing for these conditions would be best accomplished by Whole Genome Sequencing.[107]

52. Importantly, in offering an opinion as to potentially causative alternative diagnoses in her report, Professor Ryan did not distinguish between known or recognised genetic disorders which could be identified through Whole Genome Sequencing, and as yet unknown or unrecognised genetic disorders which could not.

53. In respect of potential genetic causes, Associate Professor Fahey provided to Dr Buckley for analysis a list of 204 genes known to be associated with abnormal creatine metabolism, alternating hemiplegia of childhood, neurotransmitter disorders and genetic channelopathies causing infantile encephalopathies and cardiac arrhythmias. Associate Professor Fahey's list took into account the conditions mentioned in Professor Ryan's report, as well as other relevant genetic variants.[108]

54. When taken together with the hypothesis-free analysis undertaken by the Sydney genetics team, Associate Professor Fahey considered that the genetic investigations in relation to Patrick, his siblings and mother for an alternative diagnosis to a single hypoxic episode as raised by Professor Ryan were "comprehensive".[109] Because no relevant pathogenic genetic mutation was found, he opined that all recognised genetic conditions are now excluded as the cause of Patrick's ALTE and death.[110]

55. Professor Ryan agreed with the scope of the testing undertaken, and with Associate Professor Fahey's conclusion as to the results.[111]

56. Associate Professor Fahey considered the variability in Patrick's presentation to be an issue warranting consideration.[112] However, in contrast to Professor Ryan, he

[106] Exhibit AJ, Expert report of Professor Monique Ryan (15 March 2019) pp 13, 15.
[107] Exhibit AJ, Expert report of Professor Monique Ryan (15 March 2019) pp 13, 15.
[108] Transcript of the Inquiry, 17 April 2019 T588:15-30; Exhibit AK, Expert report of Associate Professor Michael Fahey (30 March 2019) p 4.
[109] Exhibit AK, Expert report of Associate Professor Michael Fahey (30 March 2019) pp 4, 16.
[110] Exhibit AK, Expert report of Associate Professor Michael Fahey (30 March 2019) pp 4, 16; Transcript of the Inquiry, 17 April 2019 T588.24.
[111] Transcript of the Inquiry, 17 April 2019 T583.6-43.

concluded that Patrick's presentation and his pathology at post-mortem were consistent with a severe hypoxic event on 18 October 1990.[113] He noted that no alternative diagnosis had been found.[114]

57. Associate Professor Fahey identified the following material available at the time of trial which evidenced a hypoxic episode on 18 October 1990:[115]

    a. Patrick's oxygen saturation on presentation of 88 per cent.

    b. That Patrick was poorly responsive to painful stimuli and glycosuria.

    c. Dr Wilkinson's evidence that it is:

        Quite common in asphyxiation to find that there's effectively a honeymoon period that the child is brought in and there is a period of hours or days when there seems to be recovery and no major neurological problem, and subsequently they develop particularly seizures.[116]

    d. Dr Dezordi's evidence that "objectively, there is no doubt that Patrick was hypoxic when he came in, because the blood saturation tests proved that. There was no question that he was hypoxic".[117]

58. Professor Ryan was not briefed with Dr Wilkinson's or Dr Dezordi's oral evidence from the trial when she prepared her report.[118] Associate Professor Fahey and Dr Colley agreed that Patrick was hypoxic on the basis of the 88% oxygen saturation reading.[119] Professor Ryan did not accept that by reason of the oxygen level reading of 88% tPatrick was hypoxic, because an oximetry probe, as was used, can "misread" blood oxygen levels and because the reading was "low but not terribly low".[120] However, Associate Professor Fahey pointed additionally to the ambulance officer's specific observations that Patrick had poor respiratory effect, referring to a reduced drive to take breaths, which can signify the cause

[112] Exhibit AK, Expert report of Associate Professor Michael Fahey (30 March 2019) p 12.
[113] Transcript of the Inquiry, 17 April 2019 T595.27-31.
[114] Transcript of the Inquiry, 17 April 2019 T595.27-31.
[115] Exhibit AK, Expert report of Associate Professor Michael Fahey (30 March 2019) p 8.
[116] 23 April 2003 T874.53-58.
[117] 9 April 2003 T452.10-22.
[118] Exhibit AJ, Expert report of Professor Monique Ryan (15 March 2019), letter of instruction.
[119] Transcript of the Inquiry, 17 April 2019 T592.5-29.
[120] Transcript of the Inquiry, 17 April 2019 T585.34-T586.2.

being related to the brain rather than anywhere else, such as a cardiac condition or obstruction of the airway.[121]

59. By reference to literature concerning presentation of children with hypoxia, Associate Professor Fahey also identified other instances of seizures beginning after initial presentation followed by "a striking interval of near normality before neurological deterioration" with an evolution of the seizure disorder in some instances over days.[122] He said he was "satisfied from that that this was a possibility after hypoxia and that it had been reported, in fact remarkably similar to how Patrick presented".[123]

60. Associate Professor Fahey also noted that the changes seen on Patrick's brain on the CT scans proximate to his presentation on 18 October 1990 most likely represented those of hypoxia-ischemia, given what was observed at post-mortem.[124] Professor Kirk agreed that the post-mortem pathology was consistent with a hypoxic event.[125]

61. Associate Professor Fahey explained that he found it "very difficult to walk away from" the post-mortem pathology findings of brain damage, with no other mechanisms of brain damage except for ischaemic changes meaning Patrick had hypoxia at some stage, in circumstances where there was a sentinel event occurring on 18 October 1990 with emerging ischaemic CT changes from that time,[126] and no other subsequent seizures being associated with a period of hypoxia.[127]

62. Associate Professor Fahey affirmed Dr Cala's reservations about attributing Patrick's ALTE and subsequent death to a degenerative neurological condition. He did not accept the suggestion that Patrick had a "deteriorating" or "progressive" condition and preferred the term "evolving". He explained "we're not finding anything suggestive, either pathologically, biochemically, or genomically, that suggests that he had a, an underlying progressive disease." He also noted that the

[121] Transcript of the Inquiry, 17 April 2019 T592.18-25, T602.44-48.
[122] Exhibit AK, Expert report of Associate Professor Michael Fahey (30 March 2019) p 13; Transcript of the Inquiry, 17 April 2019 T593.13-15.
[123] Transcript of the Inquiry, 17 April 2019 T593.21-22.
[124] Exhibit AK, Expert report of Associate Professor Michael Fahey (30 March 2019) p 15.
[125] Transcript of the Inquiry, 17 April 2019 T600.49-50.
[126] Transcript of the Inquiry, 17 April 2019 T603.45-50.
[127] Transcript of the Inquiry, 17 April 2019 T606.14-15.

changes seen in Patrick's brain pathology were "old… not… active, not changes where the cells would deteriorate."[128]

63. Professor Ryan accepted the possibility that Patrick did have a single hypoxic ischaemic episode on 18 October 1990, but not that this was a reasonable possibility.[129] Even after having received the geneticists' reports, and heard Associate Professor's Fahey's, Dr Colley's and Professor Kirk's opinion about Patrick's hypoxic presentation and his post-mortem brain pathology, Professor Ryan maintained there was nothing that caused her to change her opinion "at all".[130] In maintaining her opinion, Professor Ryan relied on the following matters in addition to Patrick's variability:[131]

    a. The possibility of an alternative diagnosis of an unknown genetic cause, as yet unrecognised by the field of genetics.[132]

    b. A suggestion, by reference to a paper which Associate Professor Fahey referred to, that when children or adults have a severe hypoxic ischaemic injury that there is evidence of other organ injury after the fact, such as kidney failure.[133]

    c. Disagreement with the suggestion in Associate Professor Fahey's report that where there is an unrecognised first epileptic seizure on presentation there is a family history.[134]

64. In respect of Professor Ryan's possible alternative diagnosis of an unidentified genetic cause, Associate Professor Fahey accepted the possibility that in the future a genetic cause of Patrick's presentation, unrecognised as at April 2019, may become recognised.[135]

65. As an example of a "potential alternative" unidentified genetic cause for Patrick's presentation on 18 October 1990 and his subsequent course and associated findings, Professor Ryan referred to children with a clinical presentation consistent

[128] Transcript of the Inquiry, 17 April 2019 T608.44-45.
[129] Transcript of the Inquiry, 17 April 2019 T587.12-21.
[130] Transcript of the Inquiry, 17 April 2019 T587.17-21; T597.34-T598.5.
[131] Transcript of the Inquiry, 17 April 2019 T587.35-41.
[132] Transcript of the Inquiry, 17 April 2019 T584.44-47; T587.17-21.
[133] Transcript of the Inquiry, 17 April 2019 T599.17-T600.16; J E Constantinou et al 'Hypoxic-ischaemic Encephalopathy After Near Miss Sudden Infant Death Syndrome (1989) 64 *Archives of Disease in Childhood* 703.
[134] Transcript of the Inquiry, 17 April 2019 T597.41-T598.1.
[135] Transcript of the Inquiry, 17 April 2019 T590.10.

with Dravet syndrome, but without the recognised associated genetic mutation (SCN1A). Dravet syndrome involves prolonged seizures in the context of fever (especially significant fever according to Professor Ryan),[136] resulting in hypoxic ischaemic brain injuries of the sort seen on Patrick's brain post-mortem.[137]

66. Associate Professor Fahey agreed "the seizure threshold is lowered by having a fever".[138] He also agreed there are other as yet unrecognised genetic causes associated with the Dravet syndrome clinical presentation. He emphasised however, most significantly, that the presentation necessarily involved being hypoxic on presentation, which was not otherwise accepted by Professor Ryan, and also that such presentation typically presents with fitting movements of the body, which were not observed in Patrick on 18 October 1990.[139]

67. In relation to the suggestion of organ injury ordinarily following severe hypoxic ischaemic injury, Associate Professor Fahey responded that the paper referred to by him had an inclusion criteria of "or" not "and" in respect of organ failure and additionally that the paper included people "just like Patrick".[140] Professor Kirk noted that he did associate organ damage with hypoxic events, but that was based on his experience with newborn babies and noted there are special circumstances at the time of birth that do not apply to four month old children.[141]

68. In respect of Professor Ryan's possible alternative diagnosis of unrecognised first epileptic seizure on presentation,[142] Associate Professor Fahey additionally observed that if this was the case, the first seizure was very different from any other seizure that Patrick presented with across his life, which he considered unusual.[143] Dr Colley considered it relevant that in this family, there were three other children who also died young without any evidence of epilepsy or seizure, which is inconsistent with a genetic epileptic encephalopathy in the family.[144]

[136] Transcript of the Inquiry, 17 April 2019 T604.5-19, T604.47-T605.1.
[137] Transcript of the Inquiry, 17 April 2019 T604.11-13.
[138] Transcript of the Inquiry, 17 April 2019 T605.5-6.
[139] Transcript of the Inquiry, 17 April 2019 T605.8.
[140] Transcript of the Inquiry, 17 April 2019 T600.11-15.
[141] Transcript of the Inquiry, 17 April 2019 T601.1-8.
[142] Transcript of the Inquiry, 17 April 2019 T597.45-49.
[143] Transcript of the Inquiry, 17 April 2019 T593.38-46.
[144] Transcript of the Inquiry, 17 April 2019 T600.25-28.

Submissions on cause of Patrick's ALTE

69. As with Caleb, there have been two material changes since the 2003 trial. First, genetic testing has been completed and no genetic variant which is pathogenic or likely pathogenic has been identified to account for Patrick's ALTE. Secondly, more recent research on SIDS that maternal smoking and sleeping position pose the highest risks relevantly reduces any assessment of Patrick's risk of SIDS or ALTE.

70. The medical experts gave broadly consistent evidence at the trial that the ALTE was most likely caused by an asphyxiating event.

71. Before the Inquiry, Professor Ryan was not "convinced" that this was the case, whereas Associate Professor Fahey expressed the opinion that Patrick's presentation was consistent with a severe hypoxic event on 18 October 1990.

72. Professor Ryan's opinion as to Patrick's presentation and subsequent variability not being consistent with a single hypoxic episode on that date should be rejected. The opinion is mere conjecture. Moreover, it is inconsistent with the opinion evidence of multiple other relevantly qualified witnesses at the trial and in the Inquiry, the foundation for which is found in clinical records of Patrick's presentation and medical history prior to and after the ALTE.

73. When asked in oral evidence whether the consensus opinion of the other expert witnesses affected her initial opinion, Professor Ryan responded that it did not. Further, she did not accept the uncontroversial proposition grounded in lay and medical evidence that Patrick was a healthy and normally developing baby immediately prior to the ALTE.

74. In addition, Professor Ryan impliedly accepted in any event the reasonableness of the proposition that Patrick suffered a single hypoxic episode on that date by suggesting prolonged seizure in the context of fever resulting in ischaemic damage, akin to Dravet syndrome, as a potential alternative unidentified genetic cause of the ALTE.

75. We submit that the Judicial Officer should be satisfied on the medical evidence that Patrick sustained a single hypoxic event or asphyxiating event on 18 October 1990.

76. On the basis of the medical evidence, both clinical and expert, there is no identified natural cause of the ALTE, in the sense of something more than a

debating point possibility. In particular, that evidence does not support as the cause of Patrick's ALTE a respiratory problem or neurological condition such as encephalitis including a degenerative neurological disease, or a SIDS-type event.

77. The medical evidence does not exclude that the ALTE was caused by an asphyxial event including smothering. Expert opinion evidence supports an asphyxial event having occurred, with a cause other than one attributable to a respiratory or a recognised neurological condition.

78. Ultimately, on the medical evidence in 2019 there remains no identifiable natural (including genetic) cause of Patrick's ALTE and that it occurred from unnatural causes cannot be excluded.

Patrick's death

Overview of evidence on cause of Patrick's death

79. Dr Marley, Patrick's GP gave evidence that:

> *Patrick was progressing well and growing well... Patrick was no different from many other children. We saw him for minor respiratory infections… Most infants have about six viral incidents a year on average. This child had less.*[145]

80. The only differences in Patrick were his visual problems and seizure disorder.[146]

81. A record made by Dr Colley when consulting with Mr and Ms Folbigg after Patrick's death noted that the night before Patrick's death on 13 February 1991 he had a raised temperature, was sweating, vomiting and clinging.[147] However, contemporaneous hospital notes record that the night before he may have had a seizure and had a mild temperature but otherwise had "no problems".[148]

82. On 13 February 1991, one ambulance officer who attended said Patrick was not breathing at all, and another said he appeared not to be responding to CPR. A

[145] 11 April 2003 T539.13-14, T539.31, T539.55-56.
[146] Exhibit H, Forensic pathology tender bundle, p 104.
[147] Exhibit T, Expert report of Professor Cecelia Blackwell (5 March 2019), Annexure G.
[148] Exhibit S, Section of Patrick's medical records p 507.

third recorded that he was slightly blue around the lips, with shallow breathing.[149] The officers applied oxygen therapy en route to hospital.

83. In hospital, Dr Wilkinson examined Patrick during resuscitation attempts. Dr Wilkinson knew that Patrick suffered from epilepsy, and felt he could have experienced an epileptic fit resulting in obstruction of his airways, because Patrick's appearance was consistent with asphyxiation.[150] Patrick's body was warm.[151] At the time, Dr Wilkinson considered that it was quite possible that an epileptic seizure, itself caused by encephalopathic disorder, had caused asphyxiation.[152]

84. Patrick's death certificate states "asphyxia due to airway obstruction (one hour)" and "epileptic fits (four months)".[153] At trial Dr Wilkinson, who signed the death certificate, said that in the absence of other medical findings asphyxia was recorded although what specifically caused the asphyxiation was never found.[154] The post-mortem showed no evidence of things that might be associated with asphyxiation, such as vomit.[155]

85. The histology report for Patrick stated that the culture for viruses and viral antigens was negative in Patrick's nasopharyngeal aspirate.[156] However, his "post mortem blood cultures grew mixed cocci and bacilli identified as E.coli, Enterococcus faecolis and Enterococcus avium."[157] It was recorded by the forensic pathologist that "these findings are not significant and probably reflect contamination. Post mortem lung tissue cultures were negative for organisms. Post mortem lung tissue cultures for viruses and mycoplasma were negative."[158]

86. The hospital-based pathologists who conducted the autopsy could not find a cause of death, but this did not exclude a seizure.[159] They excluded infective disorders, metabolic disorders that they could think of, and genetic disorders.[160]

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[149] Exhibit H, Forensic pathology tender bundle, p 49; 9 April 2003 T442.25-26.
[150] Exhibit H, Forensic pathology tender bundle, p 62.
[151] 10 April 2003 T511.17-18.
[152] 10 April 2003 T511.46-512.8; 23 April 2003 T863.9-35, T865.1-8, T881.57-882.3 (Dr Wilkinson explained encephalopathic disorder to be a disorder in which there is some abnormality within the brain); 24 April 2003 T930.8-11, T931.10-13.
[153] Exhibit H, Forensic pathology tender bundle, p 36; Transcript of the Inquiry, 20 March 2019 T158.33-T159.3.
[154] 10 April 2003 T511.27-38; Trial Exhibit 5 (Defence), Medical certificate of cause of death of Patrick (14 February 1991).
[155] 10 April 2003 T511.38-40.
[156] Exhibit H, Forensic pathology tender bundle, p 38.
[157] Exhibit H, Forensic pathology tender bundle, p 47.
[158] Exhibit H, Forensic pathology tender bundle, p 47.
[159] 11 April 2003 T560.56-58.

87. Nor did Dr Alex Kan, the senior pathologist who examined Patrick's brain, find new damage to explain the death.[161] Old damage was evident in the form of infarcts and gliosis mostly in the form of old laminar necrosis, most severe in the parieto-occipital area, which themselves were consistent with an earlier asphyxial episode.[162] The occipital part of the brain, at the back, governs the recognition of vision.[163] The distribution of lesions was unusual for herpes simplex encephalitis and certainly appeared far more likely to be the result of the ALTE.[164] Scarring could cause epilepsy.[165] Nothing was seen that could account for Patrick's death such as new injury or damage or deterioration.[166] The underlying cause of the encephalopathy was not determined.[167]

88. The autopsy of Patrick revealed nothing in the external presentation to indicate that there had been any trauma, and there was no evidence of bruising. The pathologists looked for signs of manual asphyxia such as petechia and changes in the airways and found none.[168]

89. At trial, Dr Beal and Professor Berry both said that Patrick's death could have been caused by a seizure disorder, but Professor Herdson said this was highly unlikely.[169] Professor Busuttil considered that Patrick's brain condition could have given rise to serious convulsions causing death.[170] Professor Byard said that he could not exclude epilepsy and in isolation, would have given this as the cause of death.[171]

90. Professor Berry noted in his recitation of Patrick's histology report that the organisms were thought to reflect contamination. He did not offer a different opinion in his report.[172] Professor Herdson agreed with Professor Berry.[173]

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[160] 11 April 2003 T559.46-52.
[161] 11 April 2003 T560.15-36, T563.38-40; 23 April 2003 T865.35-41; 24 April 2003 T926.29-36, T930.56-T931.3.
[162] Trial Exhibit AD, Histopathology report of Patrick (24 June 1991).
[163] 11 April 2003 T560.1-10.
[164] Trial Exhibit AD, Histopathology report of Patrick (24 June 1991).
[165] 11 April 2003 T563.35.
[166] 11 April 2003 T560.36-41.
[167] Exhibit H, Forensic pathology tender bundle, pp 37-42, 46-47.
[168] 11 April 2003 T561.37-49.
[169] 5 May 2003 T1147.38-42, T1139.20-25 (Dr Beal); 1 May 2003 T1044.1-10, T1048.31-52 (Professor Herdson), T1061.53-1062.2 (Professor Berry), T1073.52-1074; T1076.24-3 (Professor Herdson).
[170] Exhibit H, Forensic pathology tender bundle, p 311.
[171] Exhibit H, Forensic pathology tender bundle, pp, 5, 7; 7 May 2003 T1238.23-T1240.41.
[172] Exhibit H, Forensic pathology tender bundle, p 293.
[173] Exhibit H, Forensic pathology tender bundle, p 274.

91. Dr Cala, Dr Beal, Professor Herdson and Professor Berry all considered that the death could have been the result of asphyxiating event.[174] Professor Ouvrier said that it appeared to be an asphyxia episode without clear explanation.[175] Professor Busuttil stated that the death should not be attributed to asphyxia, but gave no alternative, saying only that the brain condition "could" have given rise to convulsions causing death.[176] At trial, four forensic pathologists – Dr Cala and Professors Herdson, Berry and Byard – all said they would give Patrick's death as undetermined.[177] Dr Cala maintained this view in the Inquiry.[178]

92. In the Inquiry, Professor Cordner and Professor Duflou each attributed Patrick's death to the consequences of the encephalopathic disorder he suffered, with Professor Cordner attributing it to epileptic seizures with no evidence of the underlying cause of the encephalopathic disorder, and Professor Duflou more directly attributing it to the encephalopathy brought on by the ALTE.[179] To say that encephalopathy caused the death, however, does not identify the mechanism of the final event. Professor Duflou considered that epilepsy was possibly the cause.[180] Professor Hilton described the cause of death as part of an epileptic-type illness.[181]

Contaminants?

93. All the forensic pathologists who gave evidence at the Inquiry thought the findings in relation to the post-mortem blood cultures probably reflected contamination. Professors Cordner, Hilton and Duflou gave evidence that given Patrick's autopsy was started very soon (two hours) after death, it was notable that the post mortem blood cultures showed bacteria that appeared to be gut bacteria.[182]

[174] 16 April 2003 T747.21 (Dr Cala); 5 May 2003 T1139.52-T1140.2 (Dr Beal); 1 May 2003 T1036.4-6 (Professor Herdson) T1061.53-1062.2 (Professor Berry), T1073.52-1074.9, T1076.24-3 (Professor Herdson).
[175] Exhibit H, Forensic pathology tender bundle, p 5.
[176] Exhibit H, Forensic pathology tender bundle, p 310.
[177] 16 April 2003 T747.21 (Dr Cala); 1 May 2003 T1035.46-1036.2 (Professor Herdson) T1061.53-1062.2 (Professor Berry), T1073.52-1074.9, T1076.24-3 (Professor Herdson); Exhibit H, Forensic pathology tender bundle, p 308.
[178] Transcript of the Inquiry, 20 March 2019 T163.47.
[179] Transcript of the Inquiry, 20 March 2019 T162.32-39 (Professor Duflou); T268.1, T160.39-40; T161.19, T161.21-27, T162.4 (Professor Cordner).
[180] Transcript of the Inquiry, 20 March 2019 T162.32-39, T268.1 (Professor Duflou), T160.39-40, T161.19, T161.21-27, T162.4 (Professor Cordner).
[181] Transcript of the Inquiry, 20 March 2019 T164.3-22.
[182] Transcript of the Inquiry, 20 March 2019, T153.33-35, T154.10, T153.48-T154.1.

Time of death

94. Professor Duflou noted that the time of Patrick's death was not assessed, but he was described as having a normal skin temperature by ambulance officers.[183] Professor Duflou stated that given that the only physical description provided was that the body was warm to touch, it is entirely possible for Patrick to have died at any time from when Mr Folbigg went to work until the time at which the ambulance officers arrived, with a later time more likely than an earlier time.[184]

95. In oral evidence in the Inquiry, Professor Duflou said that he did not have a specific time that Mr Folbigg had gone to work, and accepted that not knowing what time Mr Folbigg went to work rendered his statement as to the earlier time somewhat meaningless.[185] He agreed that the time of death could not be determined with any degree of certainty.[186] His evidence that it was possible that Patrick died any time from when Mr Folbigg went to work should not be accepted.

Submissions on cause of Patrick's death

96. One material change in the available evidence since the 2003 trial is that, as with Caleb, no genetic variant which is pathogenic or likely pathogenic has been identified to account for Patrick's death.

97. In addition, the Inquiry has enabled further consideration of the role infection may have played in Patrick's death. For reasons set out later in these submissions, it is submitted that Patrick's cause of death cannot be attributed to infection.

98. No medical expert, at trial or in the Inquiry, has ruled out the possibility of a seizure having caused Patrick's death. Opinions have ranged from this being highly unlikely, or not excluded, or could have, to "would say" that epilepsy caused death. Accordingly, it is possible, but no more than possible, that a seizure caused his death.

99. Most medical experts considered that the death could have been the result of an asphyxiating event. No medical expert excluded asphyxia or smothering.

[183] Exhibit L, Expert report of Professor Duflou (13 February 2019) p 30.
[184] Exhibit L, Expert report of Professor Duflou (13 February 2019) p 30.
[185] Transcript of the Inquiry, 20 March 2019 T167.1-11.
[186] Transcript of the Inquiry, 20 March 2019 T167.22-23.

Sarah

Overview of evidence on cause of Sarah's death

100. Dr Christopher Marley was a GP who saw Sarah four times for routine reasons.[187] She appeared normal and healthy.[188] Professor Berry read all the primary documents concerning Sarah and reported:

> *At four-and-a-half months of age she was thriving and developmentally normal. When seen by her paediatrician she had a viral upper respiratory tract infection…*[189]
>
> *She was seen five times by her general practitioner and given usual childhood vaccinations and treatment for a virus infection and a fungal skin rash. On 18th August she was prescribed Flucloxacillin for a cold like illness (this was discontinued by her parents on about 26th August because of difficulty in administration) and on 26th August 1993 she was seen for a croupy cough.*[190]

101. Ms Deborah Martin, an ambulance officer who attended on 30 August 1993, arrived at about 1:30am and saw Mr Folbigg performing CPR on Sarah.[191] Sarah was cyanosed around the mouth, and had mucus and vomit in her mouth.[192] She was not breathing.[193] Ms Martin gave her adrenaline and Hartmanns fluid.[194] At approximately 2:10am officers stopped drugs and CPR because Sarah was asystolic.[195]

102. The following histology was reported:

> *Source: Lung*
>
> *Profuse Coliform*

[187] 11 April 2003 T540.6-25.
[188] 11 April 2003 T540.56.
[189] Exhibit H, Forensic pathology tender bundle, p 241.
[190] Exhibit H, Forensic pathology tender bundle, p 241.
[191] Exhibit H, Forensic pathology tender bundle, p 106; 11 April 2003 T567.3, T567.27-29.
[192] Exhibit H, Forensic pathology tender bundle, p 107; 11 April 2003 T568.3-9.
[193] Exhibit H, Forensic pathology tender bundle, p 107; 11 April 2003 T568.11-12.
[194] Exhibit H, Forensic pathology tender bundle, p 108; 11 April 2003 T568.14-19; Exhibit H, Forensic pathology tender bundle, p 141.
[195] Exhibit H, Forensic pathology tender bundle, p 108; 11 April 2003 T568.24-31.

Profuse Streptococcus, Alpha Haemolytic

Scanty Staphyolococcus Aureus

Source: Spleen

Moderate Coliforms Of 3 Colonial Types.[196]

103. In addition, it was noted that "one section of larynx shows a light mixed lymphocytic inflammatory infiltrate deep to the respiratory epithelium" and "in one section there is a light interstitial acute inflammatory infiltrate which could be seen around the occasional bronchiole".[197]

The abrasions

104. On autopsy, Professor Hilton found two tiny punctate abrasions below Sarah's bottom lip. There was a recent 1.5cm scratch on her right upper arm.[198] Professor Hilton also found an occasional petechial haemorrhage on the lungs, minor congestion and minimal oedema, and also an occasional petechial haemorrhage on the surface of the heart and on the thymus gland.[199] Both lungs showed focal areas of collapse per geographic pattern, which Professor Hilton said is commonly seen at autopsy.[200] Professor Hilton considered that features seen on Sarah tended to favour SIDS. Further, if intentional suffocation was indicated as a real possibility, Sarah's death would have been categorised as undetermined.[201]

105. At trial Professor Hilton described the abrasions as being within a centimetre or two of Sarah's lower lip and not more than a few hours old.[202] He said they were extremely superficial, to the outmost layer of skin, of a pinpoint size and were consistent with application of very minor force, which could have been by Sarah herself or by resuscitative measures.[203] He maintained this view in the Inquiry.[204] He thought it was highly unlikely that the abrasions were caused by suffocation by

[196] Exhibit H, Forensic pathology tender bundle tabs 33A, 33B.
[197] Exhibit H, Forensic pathology tender bundle, p 100.
[198] 14 April 2003 T616.34-37, T652.43-45, T652.26-31 (Professor Hilton); Exhibit H, Forensic pathology tender bundle, p 96.
[199] 14 April 2003 T618.38-T619.12, T620.4-17; Exhibit H, Forensic pathology tender bundle, p 96.
[200] Transcript of the Inquiry, 20 March 2019 T173.30.
[201] 14 April 2003 T653.38-T654.44.
[202] 14 April 2003 T617.17-50, 24 April 2003 T918.14-25; Exhibit H, Forensic pathology tender bundle, p 95.
[203] 14 April 2003 T617.52-T618.2, T652.3-56.
[204] Transcript of the Inquiry, 20 March 2019 T170.46-172.18.

a hand with a ring on it, a soft toy or a pillow.[205] Sarah could also have made the scratch herself.

106. At trial Professor Hilton said the little haemorrhages on the lungs indicated the possibility of an asphyxia mode of death, whether by an outside party or an internal form, but this happened with the majority of SIDS deaths.[206] They were not specific to smothering.[207] Similarly, the hemorrhages on the heart and thymus gland could have been from asphyxiation of some kind. It was indicative in a limited sense but was not specific to smothering.[208] Taken together, features on Sarah tended to favour SIDS.[209]

107. At the Inquiry, Professor Hilton maintained that the presence of petechiae confined to the chest cavity is supportive of a SIDS diagnosis.[210]

108. Expert evidence at trial and at the Inquiry as to the significance of the abrasions largely endorsed Professor Hilton's observation that the abrasions could have been due to resuscitation. Professors Berry and Byard at trial, and Professor Duflou in the Inquiry, held this view.[211] Dr Cala agreed, although said abrasions on children's faces were unusual, and the abrasions were one of the reasons Dr Cala said he would not give SIDS.[212] Professor Herdson considered that whether he would give SIDS as a cause of death was dependent on whether the abrasions and the scratch were obvious and apparently significant.[213]

109. Craig Folbigg and ambulance officers attempted to resuscitate Sarah.[214] In our submission, Professor Hilton's evidence, that the abrasions could have been caused by resuscitation, should be accepted.

[205] 24 April 2003 T917.51-57; 14 April 2003 T616.34-37, T652.26-31, T652.43-45; Exhibit H, Forensic pathology tender bundle, p 95.
[206] 14 April 2003 T651.25-30.
[207] 14 April 2003 T650.46-T651.30.
[208] 14 April 2003 T619.39-40; T651.20-52.
[209] 14 April 2003 T653.43-45.
[210] Transcript of the Inquiry, 20 March 2019 T174.15-16.
[211] 7 May 2003 T1216.20-31 (Professor Byard); 1 May 2003 T1063.42-51 (Professor Berry); Transcript of the Inquiry, 20 March 2019 T173.4-8 (Professor Duflou).
[212] 16 April 2003 T748.6-25.
[213] 1 May 2003 T1037.1-4.
[214] Exhibit H, Forensic pathology tender bundle, p 106; 11 April 2003 T567.21-29.

Uvula

110. Professor Hilton also found that Sarah's uvula was reddened, which at trial he said was consistent with a mild infection or snoring. He said this was also relevant to SIDS.[215] In the Inquiry he said the uvula was congested, red and bleeding a little, with microscopic evidence of inflammation. It was not elongated.[216] The inflammation on microscopic examination was consistent with a mild respiratory infection.[217] There was no evidence of a viral infection, but Professor Hilton said profuse growth of streptococcus might indicate infection, and helped explain Sarah's reddened uvula and light inflammation of larynx.[218] This would, he said, not normally be expected to contribute significantly to death.[219] In the Inquiry, Professor Hilton repeated that Sarah had minor signs of a respiratory tract infection in her lung.[220]

111. Dr Beal referred to the inflamed and displaced uvula observed by Professor Hilton and expressed the opinion that it showed a probable throat infection.[221] Professor Herdson doubted it was significant.[222]

112. Professor Byard did not exclude the uvula as playing role in the death.[223] Professor Busuttil thought that the uvula could have obstructed Sarah's airway.[224]

113. In the Inquiry, Dr Cala opined that the uvula had no bearing on cause of death.[225] Professor Duflou cited the uvula as a reason, with Sarah's age, that he would ascribe her death as Category 2 SIDS, but he said he did not know if the uvula was significant.[226] Professor Cordner considered that the uvula put Sarah in a slightly more risky SIDS category, and that while the small signs of inflammation did not come close to a cause of death, they could have made Sarah more vulnerable to SIDS.[227]

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[215] 14 April 2003 T621.35-40, T623.4-10, T654.14-28; 7 May 2003 T1182.36-45; Exhibit H, Forensic pathology tender bundle, p 95.
[216] Transcript of the Inquiry, 21 March 2019 T303.16-41.
[217] 14 April 2003 T625.41-T626.16; Exhibit H, Forensic pathology tender bundle, p 100.
[218] 14 April 2003 T627.35-T628.24.
[219] 14 April 2003 T628.26-35.
[220] Transcript of the Inquiry, 20 March 2019 T190.13-21.
[221] 5 May 2003 T1142.17-23.
[222] 1 May 2003 T1038.23-33.
[223] 7 May 2003 T1215.51-T1216.6, T1240.47-57.
[224] Exhibit H, Forensic pathology tender bundle, p 313.
[225] Transcript of the Inquiry, 20 March 2019 T176.38.
[226] Transcript of the Inquiry, 20 March 2019 T177.27-33.
[227] Transcript of the Inquiry, 20 March 2019 T177.37-43, T178.1-T179.1.

114. At trial, Dr Cala, Dr Beal and Professor Byard all gave evidence that they considered the cause of Sarah's death to be undetermined.[228] Professor Byard said undetermined with narrowing of upper airway and no death scene description.[229] Professor Berry would have given Sarah's death, in isolation, as SIDS although Sarah was of an unusual age.[230] Dr Beal would have given SIDS as an alternative cause, but noted that Sarah was outside the age range for SIDS and was found on her back.[231] Professor Herdson considered the death to be close to SIDS, but this was dependent on the punctate abrasions – if they were obvious, he would have given undetermined.[232] Dr Cala would not give SIDS, given Sarah's age and the abrasions.[233] Professor Busuttil considered that Sarah's death was the most approximate of the four to SIDS.[234]

115. When Professor Hilton saw Sarah's uvula at autopsy, after neck organs had been removed, it overlapped the epiglottis. He said in the Inquiry that neither then, nor now, could he be certain that this was not a post-mortem artefact although he said this was a "very real possibility".[235] He considered it was possible – "and I put it no more than that" – that Sarah's snoring was because the uvula was bouncing off the epiglottis or larynx.[236] In the Inquiry, he produced a paper identifying one case of death from a uvula overlapping an epiglottis, saying that this research was mildly supportive of this as a cause in relation to Sarah.[237]

Contaminants?

116. Professor Berry reported "subsequent histological, microbiological, biochemical and toxicological examination failed to give a cause for her death."[238]

117. Professor Herdson agreed with Professor Berry's histopathology and toxicologic analysis.[239]

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[228] 16 April 2003 T747.38 (Dr Cala), T748.4; 5 May 2003 T1142.3-13 (Dr Beal); 7 May 2003 T1217.2-23 (Professor Byard).
[229] Exhibit H, Forensic pathology tender bundle, pp 6-7.
[230] 1 May 2003 T1063.53-T1064.11; Exhibit H, Forensic pathology tender bundle, p 255.
[231] 5 May 2003 T1142.8-52.
[232] 1 May 2003 T1036.48-T1037.9, T1045.45-51.
[233] 16 April 2003 T747.38, T748.4.
[234] Exhibit H, Forensic pathology tender bundle, p 313.
[235] Transcript of the Inquiry, 20 March 2019 T175.42-46; T234.38.
[236] Transcript of the Inquiry, 20 March 2019 T176.7-12.
[237] Transcript of the Inquiry, 20 March 2019 T175.49-T176.5; 21 March 2019 T241.45-T242.19; T Marom et al, 'Otolaryngological Aspects of Sudden Infant Death Syndrome' (2012) 76(3) *International Journal of Pediatric Otorhinolaryngology* 311.
[238] Exhibit H, Forensic pathology tender bundle, p 255.
[239] Exhibit H, Forensic pathology tender bundle, p 274.

118. Professor Busuttil reported that "some bacteria – especially important being *Staphylococcus aureus* – were isolated from her airways at autopsy. This bacterium is not infrequently found in SIDS."[240]

119. At the Inquiry, Professor Hilton said that the findings of staphylococcus were of no significance whatsoever because it is a post-mortem contaminant, regularly found in post-mortem.[241]

120. In relation to the streptococcus he said it:

> *Might indicate that at or about or prior to the death there was a genuine streptococcul infection present in the throat or in the respiratory tract, most probably within the throat, and this again would help to explain the reddening of the uvula and perhaps the inflammation, the light inflammation in the larynx.*[242]

121. He concluded that he "would normally not have expected this degree of inflammation to have contributed significantly to this child's death".[243]

122. Dr Cala considered the profuse coliforms to be contaminants.[244] Professor Duflou's comments in relation to Patrick also applied in relation to Sarah; specimens were received on 2 September 1993 three days after her death, raising contamination as a greater possibility.[245]

Time of death

123. In his report, Professor Duflou noted that Sarah's skin temperature was described by ambulance officers as either normal or cold, and the stomach contents at autopsy were described as moderate in quantity and consisting of curdled milk with or without egg white. He stated that this would suggest that Sarah died closer to the time she was put to bed by Mr Folbigg at around 9:00pm, rather than when found by Ms Folbigg at around 1:30am. He disagreed with Professor Hilton's finding at autopsy that the time of death was 1:30am.[246]

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[240] Exhibit H, Forensic pathology tender bundle, p 313.
[241] 14 April 2003 T628.2-10.
[242] 14 April 2003 T628.18-24.
[243] 14 April 2003 T628.33-35.
[244] Transcript of the Inquiry, 20 March 2019, T186.41-43.
[245] Transcript of the Inquiry, 20 March 2019, T186.47-T187.15.
[246] Exhibit L, Expert report of Professor Johan Duflou (13 February 2019) p 33.

124. It appears that Professor Duflou was not provided with the evidence of Senior Constable Stephen Saunders, who was a police officer at the time of Sarah's death, and who interviewed Mr and Ms Folbigg. He was told that the parents again entered that room about 9:30 or 10:00pm, and Sarah was heard to be snoring, and that Ms Folbigg heard Sarah turn over in her sleep at about 12:00 or 12:30am.[247]

125. In view of evidence of when Sarah was heard or seen after she went to bed (and accepting the unreliability of opinions based on stomach content) Professor Duflou's evidence, which was little more than a hypothesis that Sarah died closer to 9:00pm, should not be accepted.

Submissions on cause of Sarah's death

126. As with Caleb, there have been two material changes since the 2003 trial. First, genetic testing has been completed and no genetic variant which is pathogenic or likely pathogenic has been identified to account for Sarah's death. Secondly, more recent research on SIDS that maternal smoking and sleeping position pose the highest risks relevantly reduces any assessment of Sarah's risk of SIDS.

127. In addition, the Inquiry has heard evidence of the role infection may have played in Sarah's death. For reasons set out later in these submissions, it is submitted that Sarah's cause of death cannot be attributed to infection.

128. The forensic pathology evidence does not identify any natural cause of Sarah's death. The only evidence of the possible role of the uvula was that it "could have" caused death or is "not excluded" as a cause.

129. Professor Cordner and also Professor Duflou referred to Sarah's reddened uvula to say that she was more vulnerable to or at higher risk of SIDS. Professor Cordner described small signs of possible inflammation, and opined that although they did not come close to a cause of death, they increased Sarah's risk of SIDS.[248]

130. However, there is no evidence other than that Sarah was a low risk of SIDS, and that SIDS itself is a rare occurrence. Relevantly, her sleep study was essentially normal. Ultimately, there remains no identified natural cause of Sarah's death.

[247] 11 April 2003 T574.57-58.
[248] Transcript of the Inquiry, 20 March 2019 T177.37-43, T178.1-T179.3, T179.25-26.

131. None of the forensic pathology or medical experts at trial excluded smothering as a possible cause of Sarah's death.[249]

Laura

Overview of evidence on cause of Laura's death

132. Testing of Laura for various diseases soon after her birth on 7 August 1997 returned normal results.[250] Dr Christopher Seton, then a physician with SIDS expertise at the Royal Alexandra Hospital for Children, conducted a sleep study which showed that Laura had mild central apnoea and no obstructive apnoea. This improved on subsequent studies and she was totally normal by February 1998.[251] The monitoring showed no serious breathing problems or heart rate problems.[252] On 17 February 1998, Dr Seton reported that Laura's sleep breathing had normalised.[253] There was no evidence of upper airway obstruction in sleep, and her sleep quality was "excellent". Correspondence from Dr Seton on 30 April 1998 recorded that her sleep breathing remained normal.[254] By April 1998, the monitoring was recorded as becoming tedious for the family and a very precautionary approach in an apparently healthy baby.[255]

133. Dr John Cash was a visiting medical officer at Singleton Hospital who examined Laura several times, including on 22 June 1998 with an upper respiratory infection and a croupy cough.[256] He found no signs of distress or respiratory difficulties, and her chest was clear, but she had mucus in her throat consistent with a cold.[257] She did not require antibiotics.[258] She attended Dr Cash's rooms on four occasions between February and May 1998, although Dr Cash did not consider this unreasonable or unusual.[259]

[249] 16 April 2003 T747.44, T749.4 (Dr Cala); 5 May 2003 T1142.10-13, 25-32 (Dr Beal); 1 May 2003 T1038.46-48 (Professor Herdson), T1065.42-44 (Professor Berry); 7 May 2003 T1217.41-45, T1256.1-15 (Professor Byard).
[250] 15 April 2003 T692.1-15; 15 April 2003 T692.1-15; Exhibit H, Forensic pathology tender bundle, p 65.
[251] 15 April 2003 T692.17-22, T693.51-54; Exhibit H, Forensic pathology tender bundle, p 65.
[252] 15 April 2003 T693.41-49.
[253] MFI 26, Sleep study report of Laura (17 February 1998).
[254] MFI 26, Letter from Dr Christopher Seton to Dr David Sanders (30 April 1998).
[255] Exhibit H, Forensic pathology tender bundle, p 66; 15 April 2003 T696.38-47.
[256] 14 April 2003 T657.1-19; Exhibit H, Forensic pathology tender bundle, p 137.
[257] 14 April 2003 T657.21-32.
[258] 14 April 2003 T657.48-52.
[259] 14 April 2003 T6660.1-22.

134. Dr Paul Innis was Laura's treating GP from 14 August 1998 until February 1999. He gave evidence at trial that Laura was normal and healthy with no chronic illness.[260] She had two episodes of croup, and flu-like symptoms on 14 August 1998 with no respiratory distress.[261] He saw her with an itchy rash, red throat and fevers in January 1999 but by February 1999 her throat was clear. He said that Laura's death was totally unexpected.[262]

135. Brian Wadsworth, an ambulance officer who attended the Folbigg home for Laura on 1 March 1999 said that when he arrived she was not breathing and had no pulse.[263] He saw no blood, vomit or foreign object inside her mouth.[264] Officers performed CPR, recorded a trace of Laura's heart with an ECG monitor and administered adrenalin.[265] Laura's skin was warm to touch.[266] She had blue colouring around the lips and face.[267]

136. Laura's histology report stated:

> *Source: Lung*
>
> *Profuse Post mortem contaminants.*
>
> *Profuse coliform.*[268]
>
> *Source: Spleen.*
>
> *Moderate coliforms of 2 colonial types.*
>
> *Profuse alpha haemolytic Streptococcus of 2 colonial types.*
>
> *Moderate Staphylococcus aureus.*[269]
>
> *Histological examination of tissues showed an inflammatory infiltrate in the heart, consistent with myocarditis, of probable viral origin. This*

[260] 15 April 2003 T668.12-T669.7.
[261] Exhibit H, Forensic pathology tender bundle, p 138.
[262] 5 April 2003 T668.18-669.7; Exhibit H, Forensic pathology tender bundle, p 139.
[263] Exhibit H, Forensic pathology tender bundle, pp 142-143, 156; 15 April 2003 T699.53-700.14, T700.16-19, T700.50-54.
[264] Exhibit H, Forensic pathology tender bundle, pp 143, 157 (note Mr Picton stated that the ECG registered asystole); 15 April 2003 T701.8-21, T701.34-36.
[265] Exhibit H, Forensic pathology tender bundle, pp 143, 157.
[266] Exhibit H, Forensic pathology tender bundle, pp 143, 157; 15 April 2003 T702.36-41, T701.19-21.
[267] Exhibit H, Forensic pathology tender bundle, pp 143, 157; 15 April 2003 T702.36-41, T701.19-21.
[268] Exhibit H, Forensic pathology tender bundle, tab 47C.
[269] Exhibit H, Forensic pathology tender bundle, tab 47D.

accords with the history of a cold/'flu-like illness for several days prior to the death of the child. There are a variety of causes for myocarditis, including some viruses, bacteria, fungi, some immune-related disorders, some drugs, and several other causative agents.[270]

Spleen: The appearances are of a probable viral infection. There is no evidence of malignancy. Nor are there any histological features to suggest any specific underlying viral infection.[271]

137. In relation to Laura, Professor Berry referred to the lung organisms as "profuse post-mortem contaminants" and the spleen as "mixed growth".[272] Professor Herdson agreed with Professor Berry's histopathology and toxicologic analysis.[273]

138. Professors Busuttil and Byard noted that there was a history of a recent upper respiratory tract infection.[274]

139. In the Inquiry Professor Duflou, with whom the other forensic pathologists agreed, considered the orthodox view in relation to the microbiology concerning Laura to be likely contamination in the main.[275]

Myocarditis: autopsy

140. At autopsy, Dr Cala found that on macroscopic internal examination the heart, including the myocardium on section, was normal (apart from an 8mm diameter area of haemorrhage on the posterior surface of the left atrium).[276] In the Inquiry, he confirmed he did not see anything on Laura's heart macroscopically.[277]

141. The first histological examination of tissues showed an inflammatory infiltrate in the heart. This presented as, within the myocardium, a moderately dense infiltrate of lymphocytes which had aggregated in certain areas particularly subendocardially and along the superficial surface of the myocardium, although

[270] Exhibit H, Forensic pathology tender bundle, p 169.
[271] Exhibit H, Forensic pathology tender bundle, p 175.
[272] Exhibit H, Forensic pathology tender bundle, p 250.
[273] Exhibit H, Forensic pathology tender bundle, p 275.
[274] Exhibit H, Forensic pathology tender bundle, pp 281, 313.
[275] Transcript of the Inquiry 20 March 2019 T218.43-45 (Professor Duflou), T219.3 (Dr Cala), T219.12-16 (Professor Hilton), T219.28-29.
[276] Exhibit H, Forensic pathology tender bundle, p 173.
[277] Transcript of the Inquiry, 20 March 2019, T196.42.

further sections showed large aggregates in the central area of the left ventricle. In these areas, there were large clusters of lymphocytes surrounding degenerate myocytes. Myocytolysis was present. No viral inclusions were seen. The appearance was of myocarditis, probably viral in aetiology.

142. A second and third block of heart tissue confirmed the presence of aggregates of lymphocytes in a similar distribution to those in the first histological examination of the heart.

143. Dr Cala recorded in the autopsy report that the microscopic findings were consistent with myocarditis, of probable viral origin which Dr Cala stated accorded with the history of a cold/flu-like illness for several days prior to the death. He noted that there were a variety of causes for myocarditis, including some viruses, bacteria, fungi, some immune-related disorders, some drugs, and several other causative agents. He considered that the inflammatory infiltrate was consistent with myocarditis; this may have represented an incidental finding.

144. At trial, Dr Cala explained the slides that were prepared from Laura's heart. He said that four blocks were routinely taken, and he took more sections to see how florid or otherwise the condition in her heart was.[278] He did not know specifically how many.[279] He kept a diagrammatic sketch of where in the left ventricle and right ventricle the sections were taken from.[280]

Expert evidence at trial

Dr Cala

145. Before the trial, Dr Cala wrote twice to police in relation to all of the Folbigg children's deaths. In relation to Laura, in a letter of 29 June 1999, he stated that she was too old for SIDS "and had an intercurrent illness which *might* have explained her death" (emphasis in the original).[281]

146. In his letter of 19 June 2001, Dr Cala answered questions raised by police in relation to the post mortem. He was asked first, whether he made the statement that the inflammatory infiltrate may have been an incidental finding in light of the family's history; secondly, he was asked whether he could indicate what would

[278] 16 April 2003 T757.48-52.
[279] 16 April 2003 T758.6-7.
[280] 16 April 2003 T758.20-22.
[281] Exhibit H, Forensic pathology tender bundle, p 185.

have been the result of the examination if Laura's death was being looked at in isolation. Dr Cala answered as follows:

> *The inflammatory infiltrate in the sections of heart which I examined in the case of Laura Folbigg was light in amount and patchy in distribution. There is evidence in the medical literature that this amount of inflammation could be considered of no relevance in the deaths of some children who have died as a result of, for example, choking on a foreign body or who died from motor vehicle trauma. My opinion that the inflammatory infiltrate in the heart represents an incidental finding is not based on the family history, but rather after consideration of the history provided of Laura's very sudden and most unexpected death, the post mortem findings of Laura and the histological assessment of the heart together with my own knowledge and experience of the condition of myocarditis.*
>
> *In other cases, I have seen where the death of a child or adult has been due to myocarditis, the inflammatory infiltrate has been much heavier in number and more diffuse in distribution throughout the heart, although the amount of inflammation is variable from case to case. There are often observable naked eye changes when examining the heart. These changes may consist of dilatation, flabbiness and pallor of the heart, and a "striped" appearance of the heart on cut section. There may be features at post mortem examination suggestive of heart failure. This may take the form of pleural effusions (straw coloured fluid in each pleural cavity) and ascites (fluid in the abdominal cavity). I should point out that these findings are not seen in every case and there are other causes for these findings. These changes were not present with Laura Folbigg, whose heart looked normal on naked eye inspection.*
>
> *If had examined the body of Laura Folbigg in isolation, without the knowledge I had at the time of previous infant deaths in the family, I might give the cause of death as Myocarditis. The question which has been asked of me is theoretical in nature and does not represent reality for this family i.e. there were other deaths in the family. When giving an opinion in relation to a cause of death for Laura Folbigg, I cannot ignore any known relevant family history of severe illnesses or premature deaths, either infantile or adult. This is not to say however that such information in any way need bias or prejudice my opinion, but it is information*

nevertheless which may be of relevance in assessing any possible cause of death of Laura Folbigg.[282]

147. At trial Dr Cala gave evidence that he did not believe myocarditis played any role in causing Laura's death. He stated,

> *As I said, the heart was normal to the naked eye, but my microscopic examination did reveal inflammation of the heart. Having said that, the inflammation was quite patchy and rather mild in the sense that although the inflammation existed it was of a rather low amount as opposed to other case that I've seen where the inflammation was much heavier in the heart and in other organs.*[283]

148. Dr Cala explained, that in death from myocarditis the heart "may, but not always… it may be flabby", the left ventricle in particular "may have a stripy appearance" and may "be a bit flabby" and the chamber itself "may be a bit dilated" – he found none of these in Laura's heart.[284] The absence of these symptoms did not exclude a death from myocarditis.[285]

149. Dr Cala considered it would be very unusual to have absolutely no symptoms or signs prior to death such as fever, unwellness, or aches and pain in the joints.[286]

150. At trial Dr Cala gave oral evidence of his description of the infiltrates as being moderately dense, "in other words not heavy but not light, somewhere in between".[287] He explained that in myocarditis, there is inflammation surrounding and within the muscle cells of the heart (which really constitutes the bulk of the heart).[288] He agreed it was a significant finding that there were also large clusters of lymphocytes surrounding degenerate myocytes.[289] Myocytolysis, which Dr Cala observed microscopically in Laura's heart, is destruction and death of muscle cells, which happens in myocarditis.[290]

[282] Exhibit H, Forensic pathology tender bundle, pp 211-212.
[283] 15 April 2003 T714.22-29.
[284] 15 April 2003 T714.43-55; 16 April 2003 T756.46-54, T757.9-13.
[285] 16 April 2003 T757.5-17.
[286] 16 April 2003 T756.4-12.
[287] 16 April 2003 T759.48-49.
[288] 16 April 2003 T759.52-T760.14.
[289] 16 April 2003 T760.45-55.
[290] 16 April 2003 T761.1-5.

151. Dr Cala said that most (not all) the slides showed the presence of myocarditis. But, he said, that was not to say that the amount of inflammation was florid and heavy in those sections. He said, "I've described it as moderate, and it is true, it was neither heavy nor light, but it was fairly patchy in the area that I have described."[291]

152. Asked whether he might have given the cause of death as myocarditis looked at individually, Dr Cala said he did not think he would because, although it was present,

> *the amount of inflammation was not particularly heavy. There wasn't any evidence of heart failure, the heart to the naked eye looked pretty normal, so – and not only that, there was evidence in other organs, the lungs and spleen in particular, of lymphocytes being in there as well. In other words, indicative of some viral infection.*[292]

153. He said if pushed he "might" give the cause of death as myocarditis but he would take it no further.[293] He did not believe it to be a reasonable possibility; he acknowledged that myocarditis can cause sudden death but thought this was very unlikely with Laura.[294]

154. Dr Cala said that the conduction system of the heart (the electrical pathways) was important when considering the effect of myocarditis although it remained speculative as to what effect, if any, inflammation in the conduction system might have – finding inflammation in the conduction system of itself did not necessarily indicate exactly what mode of death the person may have suffered, whether it was due to heart failure or rhythm disturbance or some other mode.[295] He did not conduct dissection of the conductive system of Laura's heart, because "there was no real indication to do that, even though I found myocarditis".[296] He said the finding of inflammation in the conduction system (or its absence) did not take him diagnostically further – if there was no inflammation in the conduction system it

[291] 16 April 2003 T761.13-15.
[292] 16 April 2003 T761.25-31.
[293] 16 April 2003 T761.50-51.
[294] 15 April 2003 T714.11-37, T719.28-37, T754; 16 April 2003 T756.14-22, T761.17-20, T761.55-57.
[295] 16 April 2003 T758.35-57.
[296] 16 April 2003 T764.32-33.

did not rule out the possibility of a fatal cardiac arrhythmia; if there was, he could not say she had suffered an abnormality of the rhythm disturbance.[297]

155. Dr Cala attached no significance to inflammation of the subendocardial infiltrate. Clusters of lymphocytes surrounding degenerate myocytes was a significant finding, however, Laura's heart did not exhibit things that Dr Cala said may be found in a death caused by myocarditis, such as flabbiness, a stripy appearance of the left ventricle in particular, dilation of the chamber; nor did Dr Cala find fluid around the lungs or in the abdomen.[298] He noted that persons who have died from totally unrelated causes (such as car accidents) have been found to have this mild inflammatory infiltrate of the heart.[299] In later evidence, Dr Cala referred to evidence in medical literature that this amount of inflammation could be considered of no relevance in the deaths of some children who had died from other causes.[300]

156. Dr Cala also gave a statement having viewed a video of Laura 24 hours before her death, exhibiting no outward sign of symptoms.[301] He said that in it, Laura "appears in quite good health. I think it is quite unlikely that she has died as a result of the effects of myocarditis."[302]

Professor Byard

157. Professor Byard gave undetermined as the cause of Laura's death because of the circumstances given the previous deaths in the family, and he could not exclude myocarditis.[303] However, in isolation he would have given myocarditis as cause of death and did not believe it could be excluded.[304]

158. Professor Byard described myocarditis *as* "a well-known cause of sudden and unexpected death in children of all ages and may be found in infants who present in a similar manner to SIDS."[305] It is most commonly caused by viruses, although there were no confirmatory viral studies available at the time of Laura's

[297] 16 April 2003 T764.33-42.
[298] 15 April 2003 T714.39-715.4; 16 April 2003 T756.40-T757.23, T760.16-55.
[299] 15 April 2003 T715.6-12.
[300] 16 April 2003 T763.26-32.
[301] Transcript of the Inquiry, 20 March 2019 T203.15-24.
[302] Transcript of the Inquiry, 20 March 2019 T203.15-24; 15 April 2003 T719.23-26.
[303] Exhibit H, Forensic pathology tender bundle, p 281.
[304] 7 May 2003 T1220.44-54, T1258.4-12; Exhibit H, Forensic pathology tender bundle, pp 281-282.
[305] Exhibit H, Forensic pathology tender bundle, pp 281, see 334.

autopsy.[306] He did not place much significance on the video of Laura taken the day before her death, because young children could have quite significant and potentially lethal disease whilst showing very few external manifestations (and a home video was not a clinical examination).[307]

159. Professor Byard commented upon seven histological slides from Laura's heart, showing eight pieces of heart muscle.[308] In each there was an inflammatory cell infiltrate with and without degeneration of heart muscle cells.[309] He stated that this "indicates established myocarditis".[310] He stated further that in myocarditis the heart is infiltrated by inflammatory cells resulting in the death of these cells, "as we see with Laura".[311]

160. Professor Byard commented that the clinical signs and symptoms of myocarditis are very variable. An affected child may have had no indication of any illness, or only very mild symptoms resembling a cold.[312] He had personally had several cases of infants and young children who had died from myocarditis with minimal or no symptoms.[313]

161. Professor Byard had undertaken a study to review all deaths in children and babies with a diagnosis of myocarditis at the Adelaide Children's Hospital over approximately 35 years from 1951-1990. From the whole of South Australia, he identified on average fewer than one child per year with myocarditis who died – it was rare.[314] In the study, he found about 32. In 16, the death had been caused by the myocarditis – thus in about half myocarditis was the cause, in about half it was incidental.[315] Five of those 16 died suddenly and unexpectedly, and three of those five had no symptoms.[316] Professor Byard agreed that in all of the children who died, the percentage who died of myocarditis unexpectedly, with no symptoms (i.e., as with Laura) was quite small.[317] He agreed that most people (adults and children) with myocarditis do not die, and that of those who do die, most have

[306] Exhibit H, Forensic pathology tender bundle, p 280.
[307] Exhibit H, Forensic pathology tender bundle, p 333.
[308] Exhibit H, Forensic pathology tender bundle, p 332.
[309] Exhibit H, Forensic pathology tender bundle, p 333.
[310] Exhibit H, Forensic pathology tender bundle, p 333.
[311] Exhibit H, Forensic pathology tender bundle, p 333.
[312] Exhibit H, Forensic pathology tender bundle, p 333.
[313] Exhibit H, Forensic pathology tender bundle, p 333.
[314] 7 May 2003 T1246.10-22.
[315] 7 May 2003 T1246.30-33.
[316] Exhibit H, Forensic pathology tender bundle, p 333; 7 May 2003 T1220, T1246-1247.
[317] 7 May 2003 T1247.39-46, T1428.21.

symptoms and do not die suddenly.[318] He also agreed that if myocarditis was the cause of Laura's death it was quite an unusual case.[319]

162. A number of similar reports could be found in the literature.[320] It was very well recognised in paediatrics and paediatric forensic pathology as at 2003 that children with myocarditis may die suddenly and unexpectedly with no symptoms or signs.[321]

163. Professor Byard stated, that myocarditis may, however, be completely incidental to the cause of death and he had had several cases where this had happened.[322] Part of the difficulty in Laura's case was that it could not be said that it definitely caused death.[323] He referred to cases he had seen of a 19 month old boy who choked on a peanut; a seven month old boy who drowned; a three month old boy who accidentally suffocated – in all of these cases, clearly corroborated death scene findings and autopsy features enabled a precise alternative cause of death to be given, but in the absence of these, the deaths would have been attributed to myocarditis.[324]

164. Professor Byard said that Laura's moderate myocarditis, which was quite well established and well spread, was the sort that he had seen in a number of sudden death cases in children.[325]

165. He considered that when Dr Bailey opined that 5-10% of people who have a cold have myocarditis, he was confusing inflammatory cells in the heart with myocarditis.[326]

166. Professor Byard did not consider Dr Cala to have been in a better position to diagnose Laura's cause of death, because the diagnosis rested on the slides from Laura's heart; on observation at autopsy of the heart itself it looked normal.[327] However, he accepted that on Dr Cala's macroscopic observations of the heart he

[318] 7 May 2003 T1248.46-56.
[319] 7 May 2003 T1248.57-3.
[320] Exhibit H, Forensic pathology tender bundle, pp 333-334.
[321] Exhibit H, Forensic pathology tender bundle, p 334.
[322] Exhibit H, Forensic pathology tender bundle, p 334.
[323] 7 May 2003 T1220.32.
[324] Exhibit H, Forensic pathology tender bundle, p 333; 7 May 2003 T1220.32-42.
[325] 7 May 2003 T1217.50-55, T1218.47-52.
[326] 7 May 2003 T1221.31-39.
[327] 7 May 2003 T1242.1-22.

did not identify symptoms of severe myocarditis – the heart did not show obvious changes.[328]

167. In a 1997 article that Professor Byard wrote for *Medicine, Science and Law* called 'Significant Coincidental Findings at Autopsy in Accidental Childhood Death', one of the incidental findings that he warned against, because it can be mistaken as a cause of death, is established myocarditis.[329] He also stated that in determining the role played in a death by inflammatory infiltrates (such as myocarditis) in sudden infant deaths, it is often difficult to determine whether it is the cause of death or incidental.[330]

168. Professor Byard made similar observations in a chapter he authored in the textbook 'Sudden Death in infancy Childhood and Adolescence', in which he referred to myocarditis with certain death of heart cells, as also being an incidental finding in some autopsies.[331]

169. Professor Byard agreed that the myocarditis could be incidental to Laura's death but did not agree with Dr Cala that it was "probably unrelated".[332] He agreed it was possible (but not distinctly possible) that if, hypothetically, Laura had been smothered, many pathologists would wrongly conclude that Laura died from myocarditis, in isolation.[333]

Other experts at trial

170. Professor Herdson viewed the slides from the post mortem. He agreed that histopathology of Laura's heart revealed a myocarditis most probably viral in origin, and agreed with Dr Cala that it was incidental to Laura's death.[334] He concurred with a finding of undetermined, and preferred Dr Cala's opinion over that of Professor Hilton, because the myocarditis was "not a roaring one", it was fairly diffuse, and there was little cell necrosis.[335] Without the necrosis, he would have been more confident in saying it was incidental.[336]

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[328] 7 May 2003 T1242.24-37.
[329] 7 May 2003 T1243.1-14; See Roger W Byard, 'Significant Coincidental Findings at Autopsy in Accidental Childhood Death' (1997) 37(3) *Medicine, Science and Law* 259.
[330] 7 May 2003 T1243.28-26.
[331] 7 May 2003 T1243.57-T1244.24; Roger W Byard, *Sudden Death in Infancy, Childhood and Adolescence* (Cambridge University Press, 2nd ed, 2004).
[332] 7 May 2003 T1245.30-32.
[333] 7 May 2003 T1245.34-45.
[334] Exhibit H, Forensic pathology tender bundle p 275.
[335] 1 May 2003 T1039.46-T1040.8; 15 April 2003 T719.43-T721.13; 16 April 2003 T749.9, T762.3-5; Exhibit H, Forensic pathology

171. Professor Berry viewed the slides and commented that the heart muscle showed a "patchy but widespread interstitial mononuclear infiltrate in the right and left ventricles. There is no definite myocyte necrosis".[337] The inflammation was quite extensive, and he considered that most pathologists would have accepted the inflammation as the cause of death, although "I was unable to convince myself of actual damage to heart muscle cells".[338] He stated that inflammatory infiltrates in the heart are quite common in the general population and probably accompany common childhood illnesses.[339]

172. In isolation, Professor Berry considered the myocarditis to be moderate, and that it provided an explanation but he was not certain – it could have been incidental.[340] He also thought it was highly possible – "indeed, probable" – that if a child who had myocarditis was subjected to an asphyxial episode, this might precipitate an abnormal beat of the heart leading to sudden death.[341]

173. Dr Bryan Bailey, a consultant cardiologist, gave evidence that it was unlikely that myocarditis accounted for the death.[342]

174. Professor Busuttil considered that myocarditis could have been the cause, but it may also have been incidental.[343]

175. Professor Hilton was present during Laura's autopsy performed by Dr Cala and he saw the histology slides of her heart. At trial, Professor Hilton gave evidence that in his view, the slides showed a fairly extensive inflammation of the heart muscle.[344] The severity was "probably about 6 – 5, 6, somewhere like that" [out of 10], so more than just moderate intensity.[345] He opined that myocarditis possibly caused Laura's death.[346]

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tender bundle, pp 167, 275, see also 135.
[336] 1 May 2003 T1040.47-50.
[337] Forensic pathology tender bundle, p 252.
[338] Forensic pathology tender bundle, p 256.
[339] Forensic pathology tender bundle, p 256.
[340] 1 May 2003 T1065.1-9, T1074.38.
[341] 1 May 2003 T1065.29-33.
[342] 5 May 2003 T1100.42-51 (note relying on Laura's autopsy report).
[343] Exhibit H, Forensic pathology tender bundle, p 314.
[344] 24 April 2003 T907.9-10.
[345] 24 April 2003 T907.17-20.
[346] 24 April 2003 T907.41-45.

176. Professor Hilton opined that Laura's myocarditis was of an intensity and a severity and a distribution which "could have" caused her death.[347] Whether it was incidental was a difficult question to answer.[348] It was highly significant, the only pathological lesion that was present that could account for the death.[349]

Inquiry

Dr Cala

177. In his report dated 26 November 2018 Dr Cala confirmed he remained of the view that myocarditis does not adequately explain Laura's death.[350]

178. In oral evidence, Dr Cala was asked whether the description of the inflammatory infiltrate as "light in amount and patchy in distribution" in his letter of 19 June 2001 to police was consistent with what he said in the autopsy report.[351]

179. Dr Cala gave evidence that he had described the infiltrate as "moderate, up to moderate" but accepted that there appeared to be a discrepancy. He explained that in areas of examination of the heart, in particular, in the left ventricle, the inflammatory infiltrate was light and patchy – in other words, small in amount, with a small number of lymphocytes aggregated around the cardiac cells. However, it was accentuated in areas, in portions in the middle of the left ventricle to put it up maximally to moderate intensity.[352]

180. Dr Cala also said that pathologists often describe things (such as infection, tumours) as being mild, moderate, severe in amount or intensity. The inflammatory infiltrate in Laura's heart was moderate in intensity at its most severe.[353]

181. Dr Cala noted that the letter to police was written two years after the final report, and he did not recall going back to the autopsy report to see what his terminology had been. He said that overall it remained his view that the inflammation was light and patchy, but there were areas where it was more severe.[354] However, he

[347] 24 April 2003 T907.43-45
[348] 24 April 2003 T907.49.
[349] 24 April 2003 T908.9-13, T913.15.
[350] Exhibit M, Report of Dr Allan Cala (26 November 2018).
[351] Transcript of the Inquiry, 20 March 2019 T198.1-12.
[352] Transcript of the Inquiry, T198.14-22.
[353] Transcript of the Inquiry, 20 March 2019 T198.14-37.
[354] Transcript of the Inquiry, 20 March 2019 T198.24-48.

acknowledged that his view was better expressed in the autopsy report than in the letter.[355]

182. In relation to the statment that he considered the inflammatory infiltrate to be an incidental finding, Dr Cala said that as to whether that opinion was based on the family history, the answer was no. It was, rather, a standalone diagnosis that he could clearly make microscopically, ignoring the fact of the other three deaths.[356]

183. In the letter to police, Dr Cala stated that by comparison to other cases where death was due to myocarditis, the infiltrate was much heavier in number and more diffuse in distribution. In the Inquiry he confirmed that this was by comparison to his findings on autopsy.[357]

184. In relation to the statement in his letter that he might, in isolation, give the cause of death as myocarditis, Dr Cala said that this was because he would be cautious about giving an unequivocal cause of death based purely on a pathological finding. He knew that myocarditis is a potentially serious condition, but would be cautious about looking at slides and without knowing anything else about the case, say that that unequivocally was the cause of death.[358] He emphasised in his letter that even though he knew of the previous deaths, he was not prejudiced to express any particular view, but his findings were determined just by looking at the material provided.[359]

185. He agreed in the Inquiry that nonetheless, Laura could have been part of a small number of children who die of myocarditis without showing any symptoms beforehand.[360] His view in the Inquiry was that Laura did not die of myocarditis but he could not positively exclude myocarditis as being the cause of death.[361] However, his view remained that it was not a reasonably possible cause of her death and was instead incidental to her death.[362]

[355] Transcript of the Inquiry, 20 March 2019 T199.9-12.
[356] Transcript of the Inquiry, 20 March 2019 T199.43-46.
[357] Transcript of the Inquiry, 20 March 2019 T200.22-32.
[358] Transcript of the Inquiry, 20 March 2019 T200.42-46.
[359] Transcript of the Inquiry, 20 March 2019 T201.4-8.
[360] Transcript of the Inquiry, 20 March 2019 T203.40-47.
[361] Transcript of the Inquiry, 20 March 2019 T200.38, T204.6-19.
[362] Transcript of the Inquiry, 20 March 2019 T200.38, T204.6-19.

Professor Cordner

186. In his report, Professor Cordner discussed an investigation into deaths from myocarditis in children under two years of age in New South Wales and Victoria since 2000, identified on the National Coronial Information System ("NCIS"), which showed a total of 39 cases.[363]

187. Twelve cases could not be used, due to insufficient information or infection found elsewhere (so were not isolated myocarditis).[364] Of the remaining 27 cases, in two, there were no known circumstances, one had an incomplete history. Thirteen had evidence of a preceding illness (e.g. URTI, lethargy, poor oral intake). Two had a second registered cause of death (atrial septal defect and encephalitis). Two cases involved macroscopic descriptions of the heart (e.g. dilated, enlarged, heavy). Three were co-sleeping with parent/s.[365]

188. Professor Cordner referred specifically to Dr Cala's evidence at trial and drew from it a number of elements that he addressed individually.

189. The first was that in Laura's case the myocarditis was patchy and mild compared to other cases where the inflammation was more marked.[366] Professor Cordner stated as to this, that he did not think the myocarditis was patchy and mild, he thought it better described as widespread and at least moderate in degree, and went on to test the difference including canvassing opinions of colleagues at the Victorian Institute of Forensic Medicine ("VIFM") (see below at paragraph 208).

190. Secondly, in relation to whether one would expect that there be macroscopic signs on autopsy if death was due to myocarditis, Professor Cordner noted that in 13 of the 27 cases identified on the NCIS, the heart was regarded as having a normal naked eye appearance.[367]

191. Professor Cordner also referred to Weber et al (2008), in which the authors identified proven myocarditis diagnosed in 28 cases of 1,516 paediatric autopsies over a 10 period (1.8%), within an age range of 10 days to 16 years, median 10 months). In 11 there was no macroscopic evidence of abnormality in the heart.[368]

[363] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 68.
[364] Exhibit Q, Expert report of Professor Stephen Cordner (undated) pp 68-69.
[365] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 69.
[366] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 76.
[367] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 78.
[368] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 78.

Sixteen (57%) presented as sudden death, five with no apparent prodromal symptoms.[369] The symptoms in 12 were varying degrees of dyspnoea and/or tachypnoea and three with diarrhoea and vomiting, one with pyrexia and another with non-specific viral symptoms.[370]

192. The authors concluded that "[m]yocarditis is a rare cause of death in infancy and childhood, and the majority of cases present as sudden unexpected deaths", and routine histological sampling of the heart is required for detection.[371]

193. Professor Cordner gave evidence that sudden and unexpected death was not all that unusual in the population of infants and toddlers dying from myocarditis, happening in about half the cases.[372]

194. In his oral evidence, Professor Cordner was taken to other aspects of Weber et al (2008). He was asked whether he accepted the authors' conclusion that myocarditis is a rare cause of death in infancy and childhood.[373] He gave evidence on this as follows:

> *FURNESS SC: Certainly. The first one is the conclusion that myocarditis is a rare cause of death in infancy and childhood, do you accept that?*
>
> *WITNESS CORDNER: Well you know, I mean if you're making a distinction between rare and uncommon, I mean I'm not sure, I mean on page 596 under "Discussion", second line, "Myocarditis is an uncommon but distinct and recognisable cause of childhood death", so they're just using the word interchangeably.*
>
> *FURNESS SC: Do you accept their conclusion; I'm referring to what their conclusion is?*
>
> *WITNESS CORDNER: Well I'm referring to what they say elsewhere in the article, which is using the word "uncommon", I accept both of them.*

[369] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 79; M A Weber et al, 'Clinicopathological Features of Paediatric Deaths Due to Myocarditis: An Autopsy Series' (2008) 93 *Archives of Disease in Childhood* 594, 594-595.
[370] M A Weber et al, 'Clinicopathological Features of Paediatric Deaths Due to Myocarditis: An Autopsy Series' (2008) 93 *Archives of Disease in Childhood* 594,595.
[371] M A Weber et al, 'Clinicopathological Features of Paediatric Deaths Due to Myocarditis: An Autopsy Series' (2008) 93 *Archives of Disease in Childhood* 594, 594.
[372] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 79.
[373] Transcript of the Inquiry, 21 March 2019 T290.15-16.

FURNESS SC: So, you accept myocarditis is a rare cause of death in infancy and childhood?

WITNESS CORDNER: Where rare means also uncommon.

FURNESS SC: Do you have some difficulty with the word rare Professor?

WITNESS CORDNER: No, I'm just--

FURNESS SC: It's their word?

WITNESS CORDNER: --wondering why you're making such an emphasis on it, I'm happy--

FURNESS SC: This is an article that you're relying on?

WITNESS CORDNER: Yes.

FURNESS SC: And that's their conclusion, that it's a rare cause of death?

WITNESS CORDNER: And that's their way of referring to the word "uncommon."

FURNESS SC: And what they add in page 598, they say, "What this study adds is that myocarditis is a rare cause of death representing around 2% of paediatric deaths referred for autopsy", and you accept that?

WITNESS CORDNER: Yes.

…

FURNESS SC: Under the heading "Discussion", which is on the same page, the second column, the first sentence is that "The findings of this study have demonstrated that histologically proven acute myocarditis is an uncommon but distinct and recognisable cause of death", is it your view that Laura had acute myocarditis?

WITNESS CORDNER: Yes.[374]

[374] Transcript of the Inquiry, 21 March 2019 T290.15-T291.32.

195. The study by Weber et al covered autopsies of children aged 0-18 years. It found that the majority of cases (54% of the 28 cases identified in the study) occur in children less than one year of age, with a median age of 10 months. Fatal myocarditis is "relatively more common" in older children, accounting for around 5% of all childhood deaths over the age of five years.[375] In the one to four year age range over the 10 year period, five children died from myocarditis (18% of the 28 cases from the 1,516 autopsies).[376]

196. Professor Cordner said he did not refer in his report to Laura having fallen within the 18% of the 28 cases, because it was not relevant to "what use I was trying to make of the data".[377] However, he agreed with the Judicial Officer in relation to the one to four age range findings, above, that myocarditis is less common in a child under the age of four years than it is for older children.[378]

197. Thirdly, in relation to whether Laura had preceding symptoms, Professor Cordner noted that Laura did have a runny nose in the couple of days prior to her death.[379] He also noted that of the 27 cases in the NCIS review, 15 had symptoms referable to a viral illness.

198. Fourthly, regarding myocarditis causing sudden and unexpected death only in a small percentage of cases, Professor Cordner noted that in the NCIS investigation, 13 of the 27 died in hospital, so were not sudden or unexpected.[380] Twelve arrived at hospital deceased.[381] There was no information for two. He stated that on this basis, it would appear that sudden and unexpected death is not all that unusual in this population of infants and toddlers dying from myocarditis.[382]

199. Professor Cordner's attention was drawn to the study done by Professor Byard (see paragraph 161 above), in which he identified a small percentage of children

[375] M A Weber et al, 'Clinicopathological Features of Paediatric Deaths Due to Myocarditis: An Autopsy Series' (2008) 93 *Archives of Disease in Childhood* 594, 596.
[376] M A Weber et al, 'Clinicopathological Features of Paediatric Deaths Due to Myocarditis: An Autopsy Series' (2008) 93 *Archives of Disease in Childhood* 594, 596
[377] Transcript of the Inquiry, 21 March 2019 T291.20.
[378] Transcript of the Inquiry, 21 March 2019 T291.37-47.
[379] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 78.
[380] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 79.
[381] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 79.
[382] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 79.

who had died suddenly and unexpectedly from myocarditis.[383] Professor Cordner did not take issue with the statement.[384]

200. He believed that the "middle of the road" conclusion in relation to Laura's death was that considered alone, most forensic pathologists would be comfortable ascribing myocarditis, and this was Professor Cordner's own view.[385] It would, however, have been acceptable and he would support a pathologist who gave the cause of death as undetermined provided that they fully canvassed the possibility that the death could be due to myocarditis "but because it was the fourth death in the particular family there could be other factors, including but not limited to homicide, at work".[386]

201. In his oral evidence, he maintained these views expressed in his written report, saying that where he had said he would support Dr Cala – who gave the cause of death as undetermined – and fully canvass possibilities given it was the fourth death, that would include natural causes and also homicide at work.[387] By the time of the fourth death, homicide would be in his mind.[388]

Professor Duflou

202. In his report dated 13 February 2019 Professor Duflou stated that in his opinion there was "without doubt myocarditis of a severity which can readily cause sudden and unexpected death".[389] He stated both that severe myocarditis can be incidental, while relatively mild myocarditis can readily cause death.[390] However, he went on to note:

> *Acknowledging that there is no other obvious cause of death in Laura, I nevertheless consider it not unreasonable to give the cause of death as UNDETERMINED in the alternative, as proffered by Dr Cala. The reason for this is the knowledge that myocarditis can be incidental to death, and the fact that three siblings died leads one to consider causes of death where*

[383] Transcript of the Inquiry, 20 March 2019 T206.35-45.
[384] Transcript of the Inquiry, 20 March 2019 T207.1.
[385] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 80.
[386] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 80.
[387] Transcript of the Inquiry, 20 March 2019 T207.13-40.
[388] Transcript of the Inquiry, 20 March 2019 T207.42-45
[389] Exhibit L, Expert report of Professor Johan Duflou (13 February 2019) p 34.
[390] Exhibit L, Expert report of Professor Johan Duflou (13 February 2019) p 35.

death is not simply due to myocarditis but that the myocarditis may have been a contributor or incidental to death in this case.[391]

203. Professor Duflou had conducted a study examining causative versus incidental myocarditis. Strong markers to differentiate the groups purely by heart examination were not found, although the heart was generally heavier and lymphocytic infiltrate more common in fatal myocarditis.[392]

204. In oral evidence Professor Duflou confirmed his view that a cause of death of undetermined was not unreasonable but said he was also "more than happy to give it as myocarditis".[393] He considered it to be possible that there was involvement by a person causing the deaths of the children and accepted Professor Cordner's opinion that because Laura's death was the fourth death, there could be other factors including but not limited to homicide.[394]

Professor Hilton

205. In his report dated 22 January 2019 Professor Hilton concluded,

Laura died with, and highly probably because of, florid myocarditis. There was no medical evidence demonstrable or demonstrated in the report of the post mortem examination to support another cause for her death.[395]

206. In oral evidence, Professor Hilton said that he did not know that he was in complete agreement with Professor Cordner. He said that he thought "very conservatively that in my opinion Laura might have died with or because of myocarditis", then said that "she may well have died of myocarditis".[396] He said that he tended to feel myocarditis over any other objective feature in Laura's death.[397] Referred back to his comments in his report ("highly probably because of"), and to his evidence at trial ("it was the only pathological lesion… that could account for her death" and that it "could possibly" have led to her death), he said,

there is no physical evidence, no pathological evidence of any other cause of death, dead she certainly is, myocarditis she certainly has, can

[391] Exhibit L, Expert report of Professor Johan Duflou (13 February 2019) p 35.
[392] Exhibit L, Expert report of Professor Johan Duflou (13 February 2019) p 42.
[393] Transcript of the Inquiry, 20 March 2019, T208.1-2.
[394] Transcript of the Inquiry, 20 March 2019, T208.21.
[395] Exhibit O, Expert report of Professor John Hilton (22 January 2019) p 2.
[396] Transcript of the Inquiry, 20 March 2019, T208.39-40.
[397] Transcript of the Inquiry, 20 March 2019, T208.48-50.

myocarditis kill, yes it can, may it well have killed her, is it the favoured diagnosis in this particular case, yes it is.[398]

207. Professor Hilton said that he would not have given "undetermined" but "I don't think it's an entirely unreasoned conclusion from what Dr Cala has told us".[399]

VIFM

208. The Inquiry received into evidence opinions provided by forensic pathologists at the VIFM and included in Professor Cordner's report.[400] In our submission, they should be afforded no weight in the Inquiry for the following reasons. First, the VIFM pathologists were only given a selection of microphotographs of the slides of Laura's heart, which were of varying resolution, and not representative of the slides. Secondly, they were provided with no information on the circumstances of Laura's death or her clinical and family history. Thirdly, their opinions were obtained by Professor Cordner after he stated his own opinion. That opinion was that he would be happy with myocarditis as the cause of death by way of contrast to the opinion given by Dr Cala (unnamed in the email) as unascertained.[401] Professor Cordner sought their comments without revealing that he would accept a finding of "unascertained".

Submissions on myocarditis

209. Dr Cala consistently acknowledged that considered in isolation, myocarditis might have caused Laura's death, in both of his letters to police, at trial and in the Inquiry. He did not consider it to be a reasonable possibility and described it as very unlikely.

210. It may be thought that the description which opened the topic in Dr Cala's second letter to the police somewhat understated the autopsy findings. Dr Cala essentially acknowledged as much in the Inquiry. However, this was not the extent of Dr Cala's comment in that letter, and at trial Dr Cala described the infiltrate as moderately dense, not heavy but not light and rather somewhere in between. He maintained that it was fairly patchy, and while most slides showed its presence

[398] Transcript of the Inquiry, 20 March 2019, T209.28-30.
[399] Transcript of the Inquiry, 20 March 2019, T209.30-210.6.
[400] Exhibit AM, Seven reports from forensic pathologists of the Victorian Institute of Forensic Medicine.
[401] Exhibit R, Letter from Professor Stephen Cordner (8 March 2019).

that did not mean it was florid or heavy. Professor Berry also described the infiltrate as patchy, but widespread.

211. From this it can be seen that Dr Cala's evidence was not, as Professor Cordner appeared to suggest in his report, simply that the myocarditis was patchy and mild compared to other cases where the inflammation was more marked. The other cases to which Dr Cala relevantly referred were cases of death which *were* due to myocarditis, on which he reasonably drew to comment on the autopsy findings in relation to Laura. Further, he did not simply say that these other cases of death due to myocarditis were more "marked". He said that the inflammatory infiltrate was much heavier in number and more diffuse in distribution throughout the heart, than what he found in Laura's case.

212. Deaths of children from myocarditis are rare. Their rareness was identified by Professor Byard at trial. Fewer than one child per year in South Australia was identified by Professor Byard in his study spanning over 35 years through to the late 1980s. The study by Weber et al (2008) also concluded that it is rare, representing around 2% of paediatric deaths referred for autopsy. The analysis of 27 cases identified from the NCIS, discussed in Professor Cordner's report, amounts to about one each in New South Wales and Victoria per year covered by the NCIS search.

213. Deaths of children from myocarditis which are sudden and unexpected are even fewer. Professor Byard, for instance, identified five over 35 years; most who have myocarditis do not die and most who do die do not die suddenly and unexpectedly.

214. Further, the research reported by Weber et al (2008) showed that death from myocarditis in the age range of one to four years is much less frequent than in babies under one year of age.

215. Professor Cordner ultimately accepted the authors' conclusion that "myocarditis is a rare cause of death, representing around 2% of paediatric deaths referred for autopsy".[402] If in his evidence, by initially eliding the words "uncommon" and "rare", he suggested a characterisation that such deaths are simply out of the ordinary, the suggestion should not be accepted. Professor Cordner was initially unprepared to acknowledge the findings of research upon which he relied to make

[402] Transcript of the Inquiry, 21 March 2019, T291.7.

good other points in his report. In any event, he did not take issue with Professor Byard's earlier findings.

216. The weight of the evidence is that Laura's myocarditis was moderate, although this description alone does not adequately capture the diffusion or clustering of the infiltrate identified on the histology. It has been recognised and accepted by medical experts at trial and in the Inquiry that moderate (and even, on Professor Duflou's evidence at least, mild) myocarditis can cause sudden unexpected death in a child.

217. That said, Laura was of an age in which the research referred to above (and ultimately accepted by Professor Cordner) demonstrated that it is particularly rare to suffer a sudden and unexpected death from myocarditis. Laura's myocarditis was not observed upon forensic naked eye examination at autopsy. There was no evidence of other organ dysfunction indicating heart failure. It was fairly diffuse; it appears that there was cell necrosis, but little of it. That is not to suggest that it could not have caused her death; its equivocality, however, has caused reasonable expert minds to differ.

218. No expert at trial or in the Inquiry has comprehensively excluded myocarditis as possibly causing Laura's death. Dr Cala was and remains of the view that myocarditis was incidental and does not adequately explain Laura's death. In that respect, there is no change in his opinion between the trial and the Inquiry.

219. At trial, Professor Herdson agreed with Dr Cala, favouring myocarditis as incidental to Laura's death. Professor Berry also thought it could be incidental. Professor Byard would not have excluded myocarditis but preferred undetermined given the context. Professor Busuttil said Laura's death "could have been" caused by myocarditis but it may also have been incidental. Dr Bailey considered it an unlikely cause.

220. Professor Hilton's view has been quite variable. His report provided to the Inquiry indicated he had significantly changed his opinion between the trial and the Inquiry, from that myocarditis "could have" caused Laura's death to being that she died "highly probably because of" florid myocarditis. He also noted there was no medical evidence which demonstrated support for another cause of death. In the Inquiry, his opinion was ultimately to the effect that undetermined is not entirely unreasoned, but myocarditis is his favoured diagnosis. This appears to be shift from his position at trial, although not as significant as first appeared from his report.

221. Professor Duflou said he would be "more than happy" with myocarditis, but would support undetermined, in the knowledge that myocarditis can be incidental to death and that "it is possible someone was involved".[403]

222. Professor Cordner considered undetermined would not be unreasonable, with myocarditis an unexceptional diagnosis. He stated in his report, and confirmed in his evidence, his view that,

> *I believe the middle of the road conclusion in relation to Laura's death is that considered alone, most forensic pathologists would be comfortable ascribing the death in similar circumstances to Laura's as being due to myocarditis. This is indeed my own view. It would have been acceptable, and I would support a pathologist who gave the cause of death as "I(a): Undetermined", but in the comments section of the report, fully canvassed the possibilities that death could be due to myocarditis, but because it was the 4th death in the particular family there could be other factors, including but not limited to homicide, at work.*[404]

223. As with other evidence in the case, the evidence given at trial about myocarditis in relation to Laura's death needs to be considered in light of the further evidence received in the Inquiry. When Professor Cordner's and Professor Duflou's opinions in particular are weighed with expert evidence at the trial, there is a degree of difference. That difference is seen by inclusion of two expert opinions that when the autopsy findings in relation to Laura are considered alone, myocarditis would be an unexceptional diagnosis or would be the cause of death in the absence of a competing cause.

224. But the degree of difference is tempered by the ultimate qualification which attended both opinions. Both were qualified as being on autopsy results alone. Neither excluded the possibility of an unnatural cause. Both considered "undetermined" would be supported and that the possibility of other factors should be considered or canvassed, including homicide in all the circumstances.

225. Overall, it may be said that there is a difference in the range of opinions on the role of myocarditis in Laura's death now, upon autopsy findings alone, as compared with the range of opinions given at trial. There is no difference, however, in expert opinion on the possibility of an unnatural cause having caused

[403] Transcript of Inquiry, 20 March 2019, T208.1-15.
[404] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 80; Transcript of Inquiry, 20 March 2019, T207.34-45.

her death. In view of the microscopic findings by Dr Cala, the analysis by forensic pathologists both at trial and in the Inquiry in relation to the autopsy findings considered alone, the express acknowledgement by every forensic pathologist who has given an opinion in relation to Laura's death of the possibility of an unnatural cause, the particular rarity of sudden and unexpected deaths of children from myocarditis and even more so in the age range of one to four years, it is submitted that myocarditis is a possible cause of Laura's death. However, there was no evidence received in the Inquiry which would elevate myocarditis as more than a possible cause.

Smothering

Generally

226. Smothering can leave signs which may be found at autopsy, but smothering can occur without such signs.[405] The pathological findings following suffocation are often completely nonspecific, or there may be virtually nothing to find. Even where smothering may be suspected, it is often impossible to distinguish between SIDS and deliberate or accidental suffocation.[406] In this respect there is no relevant difference between the evidence given in the Inquiry, and evidence at the time of trial.[407] As a consequence, in Professor Cordner's words, diagnosed smothering is "very, very unusual, rare".[408]

227. Facial signs of smothering include petechia on eyelids, cheeks, surface of eyes; damage to the fraenulum; and bruising on the inside of the lips.[409] Professor Duflou observed in this context that petechial haemorrhages are relatively uncommon in infant cases.[410] The presence of external signs may

[405] 14 April 2003 T650.46-T651.7 (Professor Hilton); Transcript of the Inquiry, 19 March 2019, T105.26-43 (all forensic pathologists agreeing).
[406] 1 May 2003 T1034 (Professor Herdson); 14 April 2003 T649.4-12, T653.30-34, T655.54-656.6 (Professor Hilton); 7 May 2003 T1222 (Professor Byard); Transcript of the Inquiry, 19 March 2019, T111.31-112.8 (all forensic pathologists agreeing).
[407] 14 April 2003 T649.4-12, T653.30-34, T655.54-656.6 (Professor Hilton); 7 April 2003 T267.56-T268.11 (Dr Springthorpe); 5 May 2003 T1136.27-48; Exhibit H, Forensic pathology tender bundle p 216; 28 April 2003 T982.14-30, T1136.27-48 (Dr Beal); 15 April 2003 T710.9-36, T713.6-16, T729.31-39 (Dr Cala); Transcript of the Inquiry, 19 March 2019, T111.31-112.8 (all forensic pathologists agreeing).
[408] Transcript of the Inquiry, 19 March 2019, T107.13-15.
[409] 14 April 2003 T650.53-65, T651.1-4 (Professor Hilton).
[410] Transcript of the Inquiry, 19 March 2019, T106.30-33.

depend on what was used.[411] Absence of any or all of these signs does not exclude the possibility of smothering.[412]

228. There may be petechial haemorrhages on the heart, lungs and thymus, although these are also non-specific and can be found in many children where SIDS is diagnosed.[413]

All four children

229. At trial, Dr Cala and Dr Beal gave opinion evidence that either all the children died in circumstances consistent with deliberate smothering, or suffocation in relation to the death of each of them could not be ruled out.[414] Professor Herdson opined that the four children "probably died from intentional suffocation."[415] Professor Berry opined that the deaths of Caleb, Patrick and Sarah were "entirely compatible with suffocation as the cause" and he could not rule out that they all were – in relation to Laura, possibly – suffocated, and he believed that was probably the cause.[416] Professor Byard said it was possible that all the deaths and ALTEs were caused by deliberate suffocation, with the difficulty being that the pathology did not really help.[417]

230. In the Inquiry, Professor Cordner said smothering could not be excluded in any of the Folbigg children (but there are good grounds for thinking that Laura, at least, was not smothered).[418] Professor Duflou could not exclude smothering in relation to any of the children, but could not include it either, there being no evidence for it.[419]

231. Dr Cala's opinion remains that there exists the possibility that each of the Folbigg children died not from natural disease but from inflicted injury, most likely in the form of smothering.[420] Dr Cala's basis for suspecting homicide in relation to the

[411] Transcript of the Inquiry, 19 March 2019, T105-108 (Professor Hilton, Professor Cordner, Professor Duflou).
[412] 15 April 2003 T710.32-36 (Dr Cala).
[413] 15 April 2003 T710.9-36 (Dr Cala); 1 May 2003 T1037.25-52 (Professor Herdson); Exhibit D, Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) pp 503-504.
[414] 16 April 2003 T749.27-33; 5 May 2003 T1138.42-48 (Caleb), T1139.58-T1140.2 (Patrick), T1142.25-28 (Sarah), T1143.31-34 (Laura), T1145.42-47.
[415] Exhibit H, Forensic pathology tender bundle, p 275.
[416] Exhibit H, Forensic pathology tender bundle, p 256.
[417] 7 May 2003 T1225.14-39.
[418] Transcript of the Inquiry, 20 March 2019, T162.12-15.
[419] Transcript of the Inquiry, 20 March 2019, T181.38, T181.42.
[420] Exhibit M, Expert report of Dr Allan Cala (26 November 2018) p 25.

children was his concern about the existence of four deaths in one family where he was not satisfied with the causes of death that had been given.[421]

232. No forensic pathologist at trial, or in the Inquiry, has excluded the possibility that each instance of death or ALTE could have been caused by smothering.

Absence of signs

233. In his report, Professor Cordner stated that the lack of facial injuries in the Folbigg children is evidence *against* a conclusion of smothering, particularly in relation to Laura, and should be regarded as having some weight.[422] In oral evidence, he said that major signs of smothering include external injuries around the nose and mouth, and internal injuries generally around the mouth (such as bruising inside lips or frenulum, and facial petechiae).[423] He agreed that, broadly speaking, whether there are signs will depend upon the force used, instrument or implement, part of the body and the time taken.[424]

234. There were no damaged frenula, facial bruises or abrasions (aside from on Sarah's chin), or petechial haemorrhages to the eyes found in the Folbigg children.[425] In relation to Laura, Dr Cala conducted a facial dissection and did not find any bruises or other injuries.[426]

235. Neither at trial nor in the Inquiry did other medical experts place negative weight as suggested by Professor Cordner upon the absence of facial injuries. At trial, Dr Cala said that the absence of petechial haemorrhaging on eyelids and around the eyes in Laura was non-specific – their absence did not exclude the possibility and could not be used to differentiate SIDS over smothering.[427] Dr Cala observed it may be very easy to smother a very young child but in a child of 19 months, it could take 20-30 seconds or longer.[428] Similarly, Dr Hilton said that the amount of force required to deliberately smother a 10 month old child with a pillow is fairly small and agreed that one would not necessarily expect to find signs.[429]

[421] Transcript of the Inquiry, 21 March 2019, T277.13-14.
[422] Exhibit Q, Expert report of Professor Stephen Cordner (undated) pp 52-53.
[423] Transcript of the Inquiry, 21 March 2019 T248.49-50, T249.1-9.
[424] Transcript of the Inquiry, 19 March 2019, T107.48-50, T108.1.
[425] Transcript of the Inquiry, 21 March 2019, T247.41-T249.24; 16 April 2003 T752.16-23 (Dr Cala).
[426] Exhibit H, Forensic pathology tender bundle, pp 173-174.
[427] 15 April 2003 T709.55-58, T710.1-36.
[428] 15 April 2003 T713.9-24.
[429] 14 April 2003 T656.20-36.

236. At trial, Professors Berry and Byard gave evidence that suffocation in young children often leaves no trace.[430] Professor Busuttil had a similar opinion.[431] Dr Beal stated that the macroscopic and microscopic examination is rarely helpful, and facial bruising or petechiae on occasion may point away from SIDS.[432]

237. Similarly, in the Inquiry, Professor Hilton said smothering may be suspected but is almost impossible to prove, Professor Duflou agreeing in respect of a significant percentage of cases.[433] Professor Hilton agreed that the frenula is quite commonly bruised, Dr Cala saying it can be bruised and torn but not agreeing it was common.[434]

238. Professor Duflou said he could not exclude smothering (in relation to Sarah), but he could not include it either.[435] He thought that in the general population, there would be a greater likelihood of there being signs of smothering in subsequent deaths because of there being, in every case, a possibility of signs.[436] Dr Cala did not agree.[437] Professor Duflou's opinion amounted to little more than conjecture and was not cogently argued or persuasive.

239. Professor Hilton said that in his limited experience of people dying from putting their heads in plastic bags, there are absolutely no signs of anything at all.[438] Dr Cala agreed, having seen quite a lot of these.[439]

240. Professor Cordner has never had a case in which he has diagnosed smothering in an infant. Indeed, he said that facial petechiae are rarely present in cases of infant smothering.[440] Internal signs such as biting a cheek are more likely in an adult.[441]

241. It is, on the other hand, clearly accepted that as a general proposition, smothering is very hard for a forensic pathologist to distinguish from SIDS. It is rarely diagnosed. It may well leave no physical signs.

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[430] Exhibit H, Forensic pathology tender bundle, p 256; 7 May 2003 T1205.43-T1206.15, T1222.12-15, T1223.28-31 (Professor Byard); 1 May 2003 T1055.42-49, T1074.16-31.
[431] Exhibit H, Forensic pathology tender bundle, p 315.
[432] 28 April 2003 T982.1-7.
[433] Transcript of the Inquiry, 19 March 2019, T111.24-38.
[434] Transcript of the Inquiry, 21 March 2019, T247-248.
[435] Transcript of the Inquiry, 20 March 2019, T181.41-42.
[436] Transcript of the Inquiry, 20 March 2019, T185.5-10.
[437] Transcript of the Inquiry, 20 March 2019, T185.46.
[438] Transcript of the Inquiry, 19 March 2019, T115.16-18.
[439] Transcript of the Inquiry, 19 March 2019, T115.25-33.
[440] Transcript of the Inquiry, 19 March 2019, T106.28-35, T106.44-50, T107.44-49.
[441] Transcript of the Inquiry, 19 March 2019, T106.33.

242. In view of all of the forensic pathology evidence on the likelihood of finding injuries or petechiae indicative of smothering upon autopsy, there is little support for Professor Cordner's opinion that the absence of facial signs weighs against a conclusion of smothering.

Terminology

243. In his 2015 report, Professor Cordner was critical of aspects of the manner in which the trial was conducted. First, he objected to the use of various terms including questions to medical experts as to whether a child had died from an acute catastrophic asphyxiating event and the phrase "consistent with".[442]

244. Secondly, he was critical of evidence given by Dr Cala as to the circumstances of the deaths.[443] Thirdly, he applied current autopsy standards to those carried out in 1989 and the 1990s.[444]

Asphyxiation

245. During the trial a number of the forensic pathologists and other expert witnesses were asked questions about the cause of death of the children and Patrick's ALTE using language of "asphyxiation".

246. In his report, Professor Cordner described the term "asphyxia" as meaningless as it provides no information as to the cause of the asphyxiating event and forensic pathologists cannot determine whether a person stopped breathing or their heart stopped.[445] Further, "asphyxia" is not a diagnosis, is not diagnosable and is not understood in a uniform way.[446]

247. His proposition appeared to be that the jury was therefore dealing with a concept central to the trial but which had no clear meaning and thus much of the forensic evidence at trial was misconceived.

248. Professor Cordner reported that he did not find terms such as "acute asphyxiating event" in a search of pathology databases, concluding that these terms are not

[442] Exhibit Q, Expert report of Professor Stephen Cordner (undated) pp 57-59.
[443] Exhibit Q, Expert report of Professor Stephen Cordner (undated) pp 55-57.
[444] Exhibit Q, Expert report of Professor Stephen Cordner (undated) pp 5, 36.
[445] Exhibit Q, Expert report of Professor Stephen Cordner (undated) pp 40, 46.
[446] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 6.

used by pathologists in formulating the cause of death.[447] He stated "whether the phrase was intended as a rhetorical flourish or ran risks of creating unjustified alarming prospects in the jury's mind is none of my business".[448]

249. A number of the experts called at trial gave evidence of their understanding of what was meant by "asphyxia", its derivatives, and its combination with adjective phrases. Those explanations consistently either directly describe, or plainly contemplate, the term to mean an event leading to obstruction of airways, some experts going further in their explanation to describe obstruction of air into the lungs and/or impairment of oxygen levels in the blood and/or to the brain.[449]

250. In our submission there is no identified particular answer given by an expert in evidence that appears to have been non-responsive because of the expert's misconception of the meaning of the term. None of the experts appear to have demonstrated in his or her evidence, confusion about the meaning of what was being asked. Some examples follow.

251. Dr Wilkinson replied "absolutely" to a question about whether damage to Patrick's brain after the ALTE was consistent with him having suffered a catastrophic asphyxiating event from unknown causes,[450] although quite possibly an epileptic seizure could have caused asphyxiation in Patrick's ALTE.[451] Patrick's death "certainly could have been" consistent with having suffered a recent catastrophic asphyxiating event from an unknown cause, which could have been smothering.[452] He discussed changes in the brains of children suffering "some asphyxial damage" and loss of visual function following "various asphyxial events".[453]

252. Dr Singh-Khaira demonstrated no confusion in agreeing that a catastrophic asphyxiating event from some unknown cause could be one of the causes of Patrick's death,[454] explaining that he was looking for any signs of manual asphyxia such as petechiae and changes in the airways (but found none).[455] He also thought

[447] Exhibit Q, Expert report of Professor Stephen Cordner (undated) pp 47-48.
[448] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 48.
[449] 9 April 2003 T449.57-450.3; 10 April 2003 T511.32-44, T514.26-515.40; 14 April 2003 T619.14-22, T651.17-52; 23 April 2003 T876.17-25; 5 May 2003 T1139.30-34.
[450] 10 April 2003 T509.52-55.
[451] 10 April 2003 T511.22-T512.15.
[452] 10 April 2003 T514.31-49, T516.41-T517.5.
[453] 10 April 2003 T510.1-18.
[454] 11 April 2003 T560.43-48
[455] 11 April 2003 T561.37-49.

it possible that a seizure led to a catastrophic asphyxiating event and ultimately to cardiac arrest.[456]

253. Professor Herdson was "quite sure" Caleb died from a sudden catastrophic asphyxiating event of unknown causes and agreed to that proposition as to Sarah's death.[457] Patrick's ALTE and death were each consistent with such an event (epilepsy could be a cause, but one would expect a history).[458]

254. Professor Byard's evidence regarding Patrick on this point was to the effect that although it would be very unusual, the death was consistent with a seizure disorder causing a catastrophic asphyxiating event, such disorder resulting from the ALTE, the initial asphyxiating event which itself resulted from Patrick stopping breathing but the cause of which was unknown.[459]

255. In our submission, there was clearly no misunderstanding at trial as to the use of this term by the expert witnesses. No complaint was made at trial as to the use of this term. Indeed, as is clear from the summary above, that term was used by the expert witness Professor Byard, called by the defence, without any demur.

256. At the Inquiry, the forensic pathologists were asked about the term asphyxia:

> *WITNESS DUFLOU: Yes I think in the end you probably end up using asphyxia in as meaningless a way as the term cardiac arrest, in that it doesn't provide any information really in terms of what happened.*[460]
>
> *…*
>
> *FURNESS SC: So the issue is why someone was asphyxiated rather than the state of asphyxiation which means you don't have enough oxygen?*
>
> *WITNESS DUFLOU: Yes, yes on its own it's to me, it's not a term that should be used, at least in the cause of death statement, you can certainly have qualifiers to that term, as an example, positional asphyxia, but on its own I don't think it serves much purpose.* [461]

[456] 11 April 2003 T562.40-T563.7.
[457] 1 May 2003 T1035.26, T1038.53.
[458] 1 May 2003 T1035.29-T1036.11, T1042.47-T1043.34.
[459] 7 May 2003 T1214.48-T1215.19, T1237.57-T1238.1, T1238.14-T1240.41.
[460] Transcript of the Inquiry, 19 March 2019, T100.35-37.
[461] Transcript of the Inquiry, 19 March 2019, T100.43-49.

…

WITNESS CORDNER: Just to make sure that everybody understands that, if the prosecutor was asking whether there was evidence that a particular medical diagnosis - catastrophic acute asphyxiating event, was present, it's an unanswerable question because asphyxia, as we've said, is meaningless and so it was a question that is empty.[462]

257. As can be seen, Professor Duflou was particularly concerned that asphyxiation was not used in a cause of death certificate.

258. Professor Cordner stated in his report that "anyone in the street" does not understand the term asphyxia as a low level of oxygen; "most people think" of it as a mechanical interference with respiration or breathing. He also noted that the term "mechanical interference may sound a little strange to the layman".[463]

259. He then properly conceded that that is an assertion on his part and not based on evidence. He stated that forensic pathologists using the term "asphyxiation" and its derivatives in various ways, is a "further source of confusion among lay readers/consumers of forensic pathology".[464]

260. He referred to the 2008 Report of the Inquiry into Pediatric Forensic Pathology in Ontario ("the Goudge report") in which he quotes: "asphyxia may be seriously misinterpreted or misunderstood".[465]

261. In our submission, there is no basis for the Judicial Officer to form the view that the use of those terms by the prosecutor may have confused the jury so as to give rise to an error of process in the trial and further that there was any error in the conduct of the trial by admitting this evidence.

"Consistent with"

262. Professor Cordner took issue with the use of the phrase "consistent with" by forensic pathologists. At Professor Cordner's request, Professor Pollanen provided a peer review report of Professor Cordner's report. Professor Pollanen also stated

[462] Transcript of the Inquiry, 19 March 2019, T102.49-T103.3.
[463] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 43, fn 43.
[464] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 44.
[465] Stephen T Goudge, *Report of the Inquiry into Pediatric Forensic Pathology in Ontario* (Ontario Ministry of the Attorney-General, 1 October 2008) 433; Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 57.

that "consistent with" simply means "not inconsistent with", and legal minds and jurors frequently misunderstand "consistent with" to imply corroboration, support or indication, and as a result, the phrase should be avoided.[466]

263. We submit that Professor Pollanen's opinion does not of itself indicate that there was in fact misunderstanding of what was meant by the phrase at trial. Additionally, there is no evidence of any such indication at trial of such misunderstanding. Finally, what was meant by the phrase was explained by defence counsel in his closing to the jury, and by the trial judge in his summing up.

264. In closing address, defence counsel explained that the phrase "consistent with suffocation" is not proof of suffocation and may also mean consistent with a natural process.[467] He emphasised the importance, when the phrase is used, "to say, hang on a moment, consistent with suffocation means that because a person or a child could be suffocated without there being any symptoms that consequently if there are no symptoms that that would be consistent with suffocation".[468] He said, when experts say "consistent with" suffocation, they are not saying there is positive proof of suffocation.[469]

265. Defence counsel noted Professor Herdson's evidence that Caleb's death was "consistent with" deliberate suffocation, but that that meant that there were no symptoms of suffocation because suffocation can occur with no symptoms.[470] Also, regarding Professor Hilton's diagnosis of Sarah's death as being "consistent with" SIDS, counsel said "we can read into that that there were no other findings which permitted him to reach any other conclusion" (that is, of smothering).[471] Evidence remained that there was no injury to Sarah and no medical proof of suffocation. It was necessary to distinguish medical proof of suffocation from the phrase "consistent with suffocation".

266. In summing up, the trial judge explained that if a condition is not specific for a cause, this:

> *simply means that the proper medical conclusion to draw is that the postulated cause could have been the cause for the condition, but not that*

[466] Exhibit C, Expert report of Professor Michael Pollanen (1 June 2015) p 2.
[467] 14 May 2003 T1389.45-50.
[468] 14 May 2003 T1389.36-41.
[469] 14 May 2003 T1389.36-41.
[470] 14 May 2003 T1412.23-29.
[471] 15 May 2003 T1494.24-26.

it must have been, or very likely or probably was, so that an opinion that a condition is consistent with a particular cause implies that it might also be consistent with another cause or causes.[472]

267. The question for the Judicial Officer is whether the use of those terms by the prosecutor may have confused the jury so as to give rise to an error of process in the trial.

268. In our submission, Professor Cordner and Professor Pollanen are merely speculating as to the effect of those terms. There is no basis for the Judicial Officer to find that any error in the conduct of the trial arose from experts being questioned using language of asphyxiation and consistency. In addition, the summing up and the defence submissions adequately addressed the use of the terminology.

Evidence of circumstances

269. Secondly, Professor Cordner was critical of Dr Cala for giving evidence as to the circumstances of the four deaths, without evidence that he personally conducted an investigation.[473]

270. This criticism is at odds with the accepted role of the forensic pathologist, as reflected in the SIDS categories, which specifically requires the circumstances of the death to be reviewed. Professor Cordner and the other forensic pathologists who gave evidence to the Inquiry all accepted that that was an intrinsic task to be performed.[474] In this case, the sources of information as to the circumstances were the police and ambulance officers as well as Mr and Ms Folbigg. It was entirely unnecessary for Dr Cala to conduct any investigation himself.

271. We note that in a footnote early in his report, Professor Cordner stated that for the purposes of his report, he regarded forensic pathology as the autopsy-based medical speciality of the investigation of deaths reported to coroners.[475]

[472] 19 May 2003 T26.
[473] Exhibit Q, Expert report of Professor Stephen Cordner (undated) pp 56-57.
[474] Transcript of the Inquiry, 19 March 2019, T73.27-39 (Professor Hilton), T75.16-20 (Professor Duflou), T76.5-15 (Dr Cala), T79.30-48 (Professor Cordner).
[475] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 7, fn 2.

272. We can only speculate that that footnote was included to enable him to voice the criticisms he did, particularly of Dr Cala, and form the conclusions he expressed, without regard to the circumstances of the particular case.

273. During his evidence, Professor Cordner was directed to page 3 of Professor Pollanen's report:

> *At page 3 do you see there's the heading, Dr Cala Is Not An Outlier? Professor Pollanen says: "The reader of the report may get the impression that Dr Cala was an outlier in his professional view." The professor states he was not. "The report," that's your report: "does not discuss that forensic pathologists in the late 20th century were embedded in a professional culture that permitted us to make conclusions about homicidal asphyxia in cases such as the Folbigg cases. Specifically, when Dr Cala's opinion is compared to that of American forensic pathologists, some would have concurred with his view and there are still echoes of this approach even today."*
>
> *Do you accept that?…*
>
> *WITNESS CORDNER: Okay, okay. Yes, I do.*[476]

274. In our submission, Professor Cordner's criticism should be rejected as should the implication that as a result there was an error in the conduct of the trial.

Autopsy standards

275. Professor Cordner stated in his report that an "unanswerable question" is what difference it would have made to the outcome had today's autopsy standard been applied to the medico-legal investigations of all four Folbigg children.[477] He referred to the 2012 Victorian Institute of Forensic Medicine Minimum Standards: Investigation of Sudden Unexpected Death in Infancy ("VIFM Standards") and 1992 National SIDS Autopsy Protocol as minimum standards and compared "content elements" between those standards and what were addressed in the autopsy reports for each of the Folbigg children's deaths. He listed the procedural and data elements that were apparently missing from all four death investigations

[476] Transcript of the Inquiry, 20 March 2019, T218.11-31.
[477] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 37.

(some were not available at the relevant time).[478] The VIFM Standards suggest that, in the absence of a satisfactory cause of death, consideration should be given to referring the family for assessment of the likelihood of an inherited abnormality of cardiac rhythm.[479]

276. In his report, Professor Cordner did not point to any particular test or assessment that he contended may have identified relevant evidence in the case, or to any particular aspect of the pathology evidence led at trial that he would contend was deficient.

277. Professor Cordner and Professor Duflou were asked about the standard of autopsies when giving evidence in the Inquiry:

> *FURNESS SC: … I think, Professor Duflou, you were asked and gave an opinion that the four autopsies were all adequately conducted by the standard at the time?*
>
> *WITNESS DUFLOU: Yes. Some were certainly done at a higher standard relevant to the time than others but my overall view was that given the time during which they were performed, the level could be described as adequate for all of them, yes.*
>
> *FURNESS SC: Thank you. Professor Cordner, I think you applied the standards of more current times against the autopsies. Is that right?*
>
> *WITNESS CORDNER: It was really a purely descriptive exercise that really I think doesn't take us to any particular place but just to show that things do develop and the level in the way of standards in the late 80s. There are standards now and here they are so this is just pathology's attempt to try and contribute to more observations and detail that may or may not be of some use in helping to unravel the enigma.*
>
> *FURNESS SC: But you're not suggesting that the Inquiry should consider any of the autopsies in a particular way given current standards?*
>
> *WITNESS CORDNER: No, I'm not. No, I'm not.*[480]

[478] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 38.
[479] Exhibit Q, Expert report of Professor Stephen Cordner (undated) p 38.
[480] Transcript of the Inquiry, 21 March 2019, T301.19-40.

278. In the absence of any specific deficiency by which it may properly appear that there is a doubt or question as to the accuracy or sufficiency of autopsy findings in this case, we submit this issue raises matters of generalised speculation, at best.